

HUMAN

Physiology

for medical students



SPECIAL SENSES



BY
MAGDI SABRY M.D

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**HUMAN
PHYSIOLOGY
FOR MEDICAL STUDENTS**

SPECIAL SENSES

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DEDICATED TO

MY TEACHERS THROUGHOUT MY LIFE, particularly the first 2 teachers, my late parents.

MY FAITHFUL LATE WIFE, the light that disappeared from life but is everlasting inside me.

MY SONS, SHERIF, AMR AND ESSAM, AND THEIR WIVES, their love gives me hope and interest in life and makes their happiness my chief goal.

My GRANDCHILDREN, AHMED, NOURHAN AND SAMA SHERIF, YASMIN AND MOHAMMED AMR, AND AYA ESSAM, the beautiful young angels that have added a new kind of happiness to my life.

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With recent advances in the field of human physiology, it has become urgent to provide an up-to-date review on the subject.

This book is provided to help medical students in understanding modern human physiology. It presents the whole subject in a brief, comprehensive, and up-to-date form.

Great effort was done to perfect such a vast subject in an easily understandable expression and in a such reasonable bulk. It includes as much simplified and clear illustrations as possible, and to maintain simplicity, they have been presented in a diagrammatic form, photographs were greatly excluded.

The major part of my gratitude should be given to all who taught me, and to my wife and children, who patiently supported me during preparation of this book.

Any suggestions, remarks or criticism will be greatly welcomed, heartily appreciated and very much considered.

I hope this book will be a real help to undergraduate medical students, as well as to postgraduates and candidates of higher degrees, in the field of human physiology.

MAGDI SABRY

Second edition :

Preface to the third edition

This new edition is original both in shape and contents. All chapters have been extensively revised and updated. New coloured illustrations are introduced and recent data are added elsewhere specially about the mechanisms of retinal functions, and many subjects have been completely rewritten.

MAGDI SABRY

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SECTION I

PHYSIOLOGY OF VISION

CHAPTER 1

INTRODUCTION

Vision is important for (a) *Identification* of objects (b) *Learning* through written speech (c) *Maintenance of equilibrium* (refer to C.N.S.).

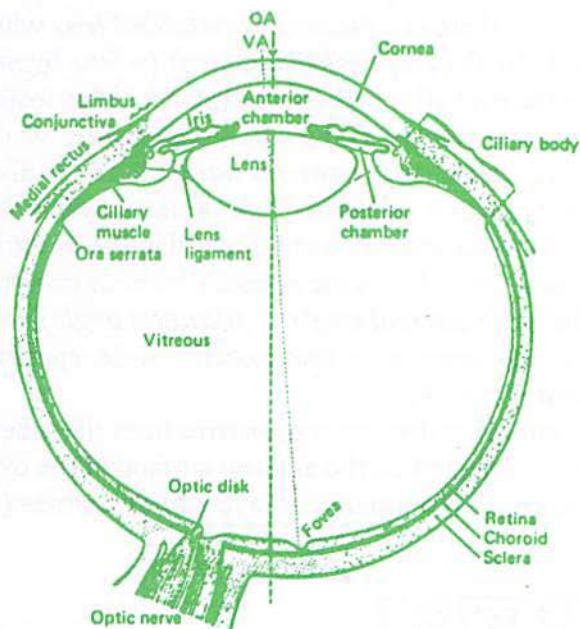


Figure 1 : A horizontal section through the right human eye.
OA = Optic Axis. VA = Visual Axis.

STRUCTURE OF THE HUMAN EYE

The adult human eyeball is nearly globular (= spherical) in shape, about one inch in diameter, and it consists of the following **3 layers** (figure 1) :

(1) **An outer fibrous (protective) layer** : The posterior 5/6 of this layer is opaque and is called the *sclera*, while its anterior 1/6 is transparent and is called the *cornea* (through which light rays enter the eye).

(2) **A middle vascular layer (the uveal tract)** : This layer is called the *choroid*. It contains many blood vessels, and its anterior extension forms the *ciliary body and the iris*. The latter is a coloured partition in front of the crystalline lens that has a central round aperture (opening) called the *pupil*.

(3) **An inner nervous layer** : This is the *retina*, which contains the *photosensitive receptors called the rods and cones*. It extends anteriorly and ends just behind the ciliary body at the *ora serrata*. In its posterior part there is a small area called the *macula lutea*, and vision is most acute in the central part of this area, which is known as the *fovea centralis*.

The site at which the optic nerve leaves the retina is called the *optic disc, optic papilla, or blind spot* (because it contains no photoreceptors).

Behind the iris, there is a *biconvex crystalline lens* which is suspended to the ciliary body by the *suspensory ligament (= lens ligament or zonule)*. The lens divides the eyeball into 2 compartments : The posterior is occupied by a gelatinous mass known as the *vitreous humour*, while the anterior is filled with a fluid called the *aqueous humour*, and is divided by the iris into two chambers called the *anterior and posterior chambers*.

The circular corneo-scleral junction is called the *limbus*, at which there is an important canal that drains the aqueous humour known as the *canal of Schlemm*. At the *irido-corneal angle (= filtration angle)*, there is a network of connective tissue trabeculae that contain wide spaces known as the *spaces of Fontana* (figure 8).

A thin membrane called the *conjunctiva* lines the inner surfaces of the eyelids, then it is reflected on the anterior surface of the eyeball, and at the limbus it continues with the superficial layers of the cornea (figure 1).

PRINCIPLES OF OPTICS

Light waves travel through air at a speed of about *300000 Km /second*. The visible wave-lengths for the human eye range from *4000 to 7250 Å* ($\text{Å} = \text{Angstrom} = 0.1 \text{ millimicron}$). Adjacent to the visible zone, there are *2 invisible zones* which include the *ultraviolet and infrared rays*.

When light rays strike a surface, they are either reflected, absorbed or transmitted through it with or without refraction. The degree of refraction depends on (a) The angle of incidence (incident rays striking the surface perpendicularly are not refracted) (b) The refractive index of the substance. The latter is the *ratio between the velocity of light rays in air and their velocity in the substance* (e.g. the velocity of rays in glass is *200000 Km / second*, so the *refractive index of glass* = $300000 / 200000 = 1.5$).

Light rays passing from air to a denser medium are refracted towards the perpendicular line, while those passing from a dense medium to air are refracted away from the perpendicular line.

THE LENSES

Concave lenses diverge parallel rays while convex lenses converge parallel rays to a focal point (= *focus*), the distance of which from the *nodal point* (= *central part of the lens*) is called the *focal distance or focal length* (*f*). The focal length depends on the power of the lens (the greater the power of the lens, the shorter will be its focal length and vice versa).

THE DIOPTER : This is the unit of the power (or strength) of lenses. The power of a certain lens in diopters equals *the reciprocal of its focal length in meters* (i.e. $1/f$) e.g. the power of a lens having a focal length 1 meter = $1/1 =$ one diopter, while the power of a lens having a focal length 0.1 meter = $1/0.1 = 10$ diopters.

REFRACTIVE MEDIA OF THE EYE

There are *4 refractive media in the eye*, which include the following :

- (1) **The cornea** : Its refractive index is 1.38.
- (2) **The aqueous humour** : Its refractive index is 1.33.
- (3) **The crystalline lens** : Its *nodal point lies at the junction of its middle and posterior thirds*, and its *central part has a greater refractive index* (and refractive power) than that of its peripheral parts (opposite to the condition in convex glass lenses). However, its average refractive index is about 1.40.
- (4) **The vitreous humour** : Its refractive index is 1.34.

There is a relation between the power of a certain lens and the *distances of both the object and its image from the lens nodal point* (in meters). Such relation is expressed by the following formula :

$$\text{Power of the lens} = (1 / \text{object distance}) + (1 / \text{image distance}).$$

In the *normal resting eye*, the nodal point of the crystalline lens lies 15-17 mm in front of the retina, and parallel rays are focused on the retina. In this case, the object distance from the eye is considered *infinity*, and by applying the above formula, *the power of the crystalline lens of the eye during rest would be 59 - 67 diopters*. However, such power is produced mainly by the cornea and not by the lens because the refractive index of air is one, and the greatest light refraction in the eye occurs at the air - cornea surface (which acts as a convex lens having a power of 39 - 43 diopters). However, *under water the cornea almost loses its refractive power because the refractive index of water (1.33) is nearly equal to that of the cornea*.

****** Although the power of the crystalline lens of the eye *in air* is about **120 diopters**, yet its power *inside the eye at rest* is only **16 -20 diopters** i.e. only about **1/3 of the overall refractive power of the eye**. This is because the lens inside the eye is surrounded by the aqueous humour anteriorly and the vitreous humour posteriorly, and these media have refractive indices that are not greatly different from that of the lens, which markedly reduces the magnitude of light refraction at the lens surfaces.

The reduced or schematic eye

This is an eye with simplified optics. In this eye, the **overall refractive power during rest** is represented by a single lens placed 15-17 mm in front of the retina, with a total power of **59 - 67 diopters** (see above).

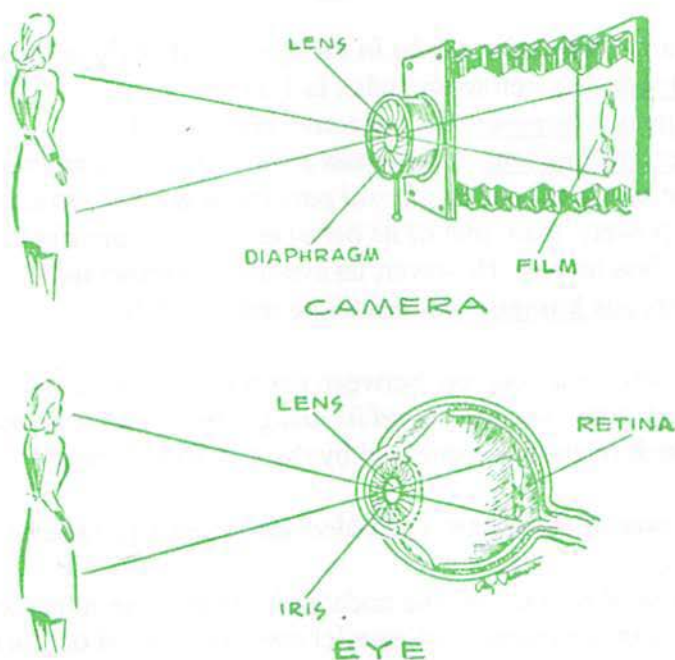


Figure 2 : The camera function of the eye (after prof. Omar A. Zaki).

The camera function of the eye

The eye functions like a camera. Parallel rays falling on the eye converge to a focus on the retina, which is analogous to the sensitive film of the camera. The iris regulates the amount of light rays admitted into the eye (by *adjusting the size of the pupil*), exactly as the diaphragm of the camera does (by adjusting the size of its central aperture). Also, the images formed on both the retina and the sensitive film of the camera are *inverted* (figure 2)

Retinal images

These are *normally inverted* (figure 2), and such position is corrected by the visual cortex. The size of these images can be calculated if the size of the object and its distance from the *nodal point of the eye lens (in meters)* are known, according to the following relation :

$$\frac{\text{Size of object}}{\text{Size of image}} = \frac{\text{Distance of object from the lens nodal point}}{\text{Distance of image from the lens nodal point}}$$

Thus, if the object size was *one meter* and its distance from the lens nodal point was 10 meters, while the retinal image distance from the nodal point was 17 mm (0.017 meter), then $\frac{1}{\text{size of image}} = \frac{10}{0.017}$. Accordingly,

$$\text{the size of the image} = \frac{0.017}{10} = 0.0017 \text{ meter} = \mathbf{1.7 \text{ mm.}}$$

The optic and visual axes

The optic axis is a straight line that joins the anterior and posterior poles of the eyeball while the *visual axis is a straight line that joins the centre of the pupil to the fovea centralis* (figure 1).

CHAPTER 2

THE OUTER LAYER OF THE EYE

(1) THE CORNEA

The cornea constitutes the *anterior 1/6 of the outer layer of the eyeball*. It is 0.5 - 1 mm thick and its diameter is about 11 mm. It performs the following functions :

- (1) It allows entry of light into the eye due to its *transparency* (see below).
- (2) It is the *most important refractive medium in the eye*, since it acts as a *convex lens having a power of 39-43 diopters* (page 3).
- (3) Its regular curvature leads to formation of *sharp retinal images*.
- (4) It is *permeable to isotonic fluids*. This property allows various kinds of eye drops to get easily into the eye.
- (5) It *protects the delicate inner structures of the eye* (with the sclera).

The cornea also protects the eye by absorption of a considerable amount of the ultraviolet rays (with the lens).

The cornea is always covered by a *thin film of tears* (which is secreted by the lacrimal glands) *that gives the eye its particular luster* (= brightness), and it also contains extremely sensitive *non-myelinated nerve endings* (which transmit pain as well as other sensations from the cornea).

It is protected by (a) The eyelids and the lid reflexes (which lead to *blinking*) (b) The precorneal film of tears (which dilutes and washes irritant substances and also contains an antibacterial enzyme called the *lysozyme*) (c) The corneal reflex (see next).

THE CORNEAL REFLEX

Touching the cornea of one eye by a foreign body (e.g. *a cotton*) causes *blinking at both eyes*. It is a *superficial reflex* (refer to C.N.S.). Afferent impulses are transmitted from the cornea via *the ophthalmic division of the trigeminal (5th cranial) nerve* to the *pons*, where they stimulate the facial (7th cranial) nuclei at both sides. Efferent impulses are then transported by *the facial nerves* and lead to contraction of the *orbicularis oculi muscles at both sides resulting in bilateral blinking*. This reflex is primarily *protective*. However, it is commonly *tested clinically* to check (a) *The integrity of the 5th nerve* (b) *The depth of anaesthesia* (see later).

Corneal nutrition and metabolism

Normally, the cornea contains *no blood vessels* (i.e. it is completely an *avascular structure*) in order to maintain its *transparency*. *Glucose* is the main source of energy. It receives its oxygen and nutrient supply from its own lymph vessels and the aqueous humour as well as from the fluid filtered from the blood capillaries at the limbus. It also contains *ascorbic acid and glutathione* (which play an important role in the oxidative processes that take place in the cornea).

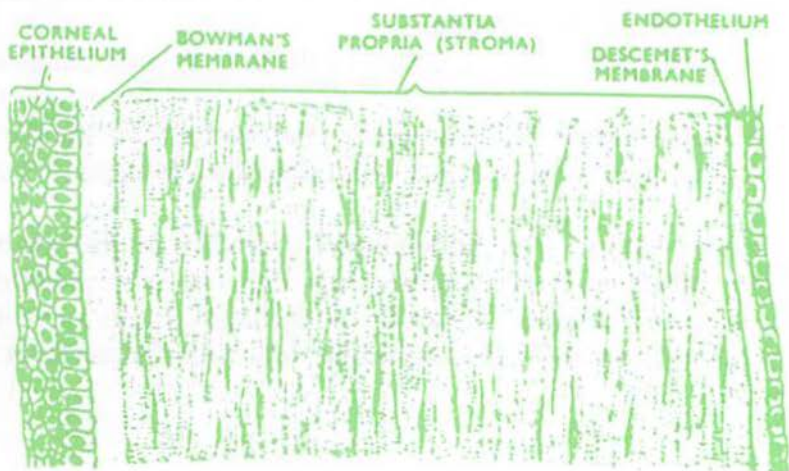


Figure 3 : Structure of the cornea.

Causes of corneal transparency

(1) **Structure** : The regular arrangement of the corneal connective tissue lamellae (figure 3) is one of the important factors for production of corneal transparency.

(2) **Avascularity** : This is the *main cause* of maintaining corneal transparency. Vascularization of the cornea often occurs as a result of deficiency of vitamin B₂ (**riboflavin**) and may lead to loss of its transparency. In addition, deficiency of vitamin A causes *xerophthalmia* (= dry eye) which may also lead to marked impairment of corneal transparency.

****** Riboflavin is essential for formation of the *flavoprotein enzyme*, which is concerned with respiration of avascular structures (e.g. the cornea and lens). Accordingly, its deficiency leads to corneal hypoxia (which is followed by its vascularization as a compensatory effect).

(3) Absence of myelin sheath in the corneal nerve fibres .

(4) Relative corneal dehydration : The transparency of the cornea also depends on its *water content*. In order to maintain the transparency of the cornea, it *should be kept relatively dehydrated* (since its excessive hydration leads to *corneal cloudiness*).

The corneal water content is determined by *an equilibrium between the processes of hydration and dehydration* in the cornea. Corneal hydration occurs as a result of flow of fluid from the blood at the limbus into the cornea (due to hypertonicity of the corneal tissue relative to the blood). On the other hand, corneal dehydration, (which is normally predominant), occurs by the following mechanisms :

(a) Metabolic pump : This is an active process that occurs in the corneal endothelial layer and results in expulsion of fluid *into the aqueous humour*.

(b) Osmotic pump : The osmolalities of the fluids that surround the cornea (the aqueous humour posteriorly and the tears anteriorly) are relatively greater than that of the cornea. Accordingly, fluids move down an osmotic gradient from the cornea outwards (which contributes to its relative dehydration).

(2) THE SCLERA

The sclera constitutes the *posterior 5/6 of the outer layer of the eye*. It is formed of *hard fibrous tissue*, and is covered anteriorly by the *conjunctival membrane* (figure 1). Normally, it is whitish in adults and bluish in infants and young children. However, its colour can be changed in disease (e.g. it becomes yellowish in jaundice). It is *opaque* (i.e. it *does not transmit light rays*) due to the *marked irregularity of its connective tissue lamellae*. However, it performs the following functions **(a)** It *protects the delicate inner eye structures* **(b)** It gives attachment to the *external ocular muscles* (which move the eyeball in various directions).

CHAPTER 3

ACCOMMODATION TO NEAR VISION

The crystalline lens of the eye

This is a biconvex transparent lens that is suspended to the ciliary body by the suspensory ligament. In young ages, its substance is malleable and its capsule is extremely *elastic* (but it becomes progressively hard with advance of age (see below). During rest, its *posterior surface has a greater curvature than its anterior surface* (figure 1). Histologically, it is formed of *concentric layers of modified fibrous cells* (with the older fibres at the centre) and chemically, it consists of about 70 % water and 30 % proteins.

Causes of transparency of the lens

- a- Absence of blood vessels and nerves. Because of its avascularity, the lens obtains its oxygen and nutrients from the aqueous humour.
- b- The uniform arrangement of the different lens fibres.
- c- The nearly equal refractive indices of the various lens constituents.

Functions of the lens

- (1) It is an important *refractive medium*, providing about 1/3 of the total refractive power of the eye optical system during rest (page 4).
- (2) Its elasticity allows it to be more spherical (with a subsequent increase in its power), which is essential for the process of *accommodation*.
- (3) It absorbs (with the cornea) a considerable amount of ultraviolet rays (thus *protecting the retina* from their harmful effects).

Metabolism of the lens

The lens has a low (but active) metabolic rate. Energy is set free by oxidation of *glucose* mainly *anaerobically to lactic acid* (which diffuses to the aqueous humour), *but some aerobic metabolism also occurs* (which leads to complete oxidation of glucose to CO₂ and water). Such aerobic metabolism is performed through the presence of (a) The flavoprotein enzyme (b) Glutathione and ascorbic acid (which act as hydrogen acceptors for the oxidation of glucose). In senile cataract (see below), the glutathione content of the lens is markedly decreased.

Cataract

This is a disease of the crystalline lens in which it loses its transparency (becoming opaque and whitish) as a result of *denaturation of its proteins*. It is a *degenerative condition* that occurs due to one of the following causes.

(1) **Exposure to ultraviolet rays** : These rays lead to denaturation of the lens proteins followed by their coagulation (forming opaque areas).

(2) **Exposure to high temperatures or x-ray radiations** : These produce similar effects as those produced by the ultraviolet rays.

(3) **Diabetes mellitus** : In this disease, cataract occurs secondary to disturbances of the glucose metabolism in the lens.

(4) **Old age (senile cataract)** : This probably occurs as a result of decreased glutathione content in the lens (see above).

****** Cataract is *treated by surgical removal of the opaque lens* and supplying the patient with convex lenses of a suitable power (in the form of either glasses, contact or implanted lenses).

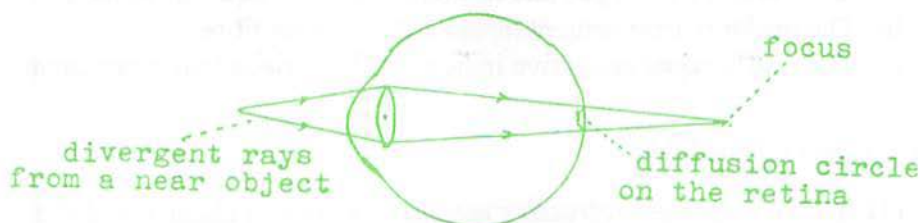


Figure 4 : Divergent rays from a near object forming a blur (or a diffusion circle) on the retina.

ACCOMMODATION (= NEAR RESPONSE OR REFLEX)

Accommodation is a process by which the optical system of the eye is adjusted to see near objects clearly. Normally, during distant vision, the ciliary muscle is relaxed, and parallel rays (= those coming from objects placed *more than 6 meters away from the eye*) are brought into a sharp focus on the retina. This *normal optical condition* is called *emmetropia*, and the optically normal eye is referred to as an *emmetropic eye* (figure 12a).

Light rays coming from objects placed at nearer distances than 6 meters from the eye are *divergent*, and they form a focus behind the retina and a blur (or a diffusion circle) on the retina (figure 4). Accordingly, these objects are indistinct, and in order to see them clearly, their images should

be brought on the retina. This is produced through the process of accommodation (= *near response or near reflex*). In this process, the lens becomes more spherical (i.e. it assumes a more convex shape), thus its power increases and brings the object's image on the retina as described below (see next).

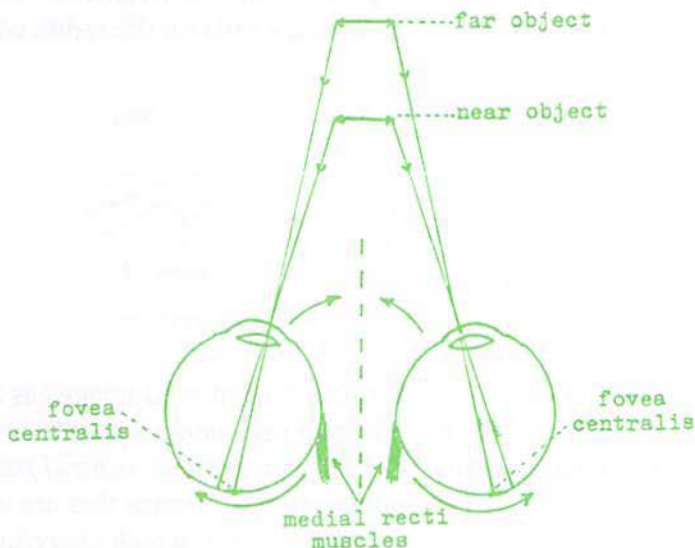


Figure 5 : Convergence of both eyes during accommodation.

Eye changes that occur during accommodation

1. Convergence of both eyes (or visual axes)

This is produced mainly by contraction of the *medial recti muscles* of both eyes, and its function is to *shift the images of the near object to the fovea centralis of both retinae* where they can be seen clearly (figure 5).

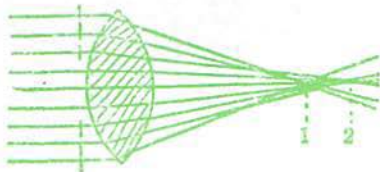


Figure 6 : Spherical aberration in a convex glass lens. Peripheral rays meet at point (1) while central rays meet at point (2).

2. Bilateral miosis (or pupilloconstriction)

This cuts off a considerable amount of the peripheral rays, thus allowing only the central rays to enter the eyes. This effect **prevents both spherical and chromatic aberrations** resulting in clear vision (see below) and also **increases the depth of focus** (i.e. the distance of the object from the eye can be changed and still its image falls on the retina without change in accommodation).

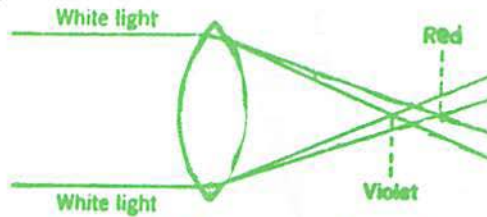


Figure 7 : Chromatic aberration.

Spherical aberration : This is formation of blurred images as a result of inability of the convex lens to collect all rays into a single focus **due to differences in the refractive powers of its peripheral & central parts** (figure 6).

Chromatic aberration : This is formation of images that are surrounded by halos of the various colours of the spectrum (which also causes blurring). This occurs as a result of **analysis of white light at the peripheral parts of the lens** (which act as **prisms**). The spectral colours are refracted by different degrees according to the wavelength of each colour (figure 7).

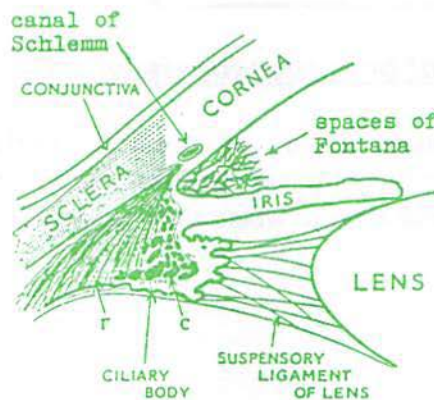


Figure 8: The iridocorneal (or filtration) angle. (r) and (c) = the radial and circular fibres of the ciliary muscle.

3. Increasing the power of the lens

During near vision, both the circular and radial fibres of the ciliary muscle (figure 8) contract, pulling the whole ciliary body forwards and inwards, thus the edges of these bodies come closer to each other. This results in *slackening (i.e. relaxation) of the suspensory ligament*, so the lens assumes a more spherical shape (due to its elasticity) and its convexity increases (figure 9). Accordingly, the lens' power increases, thus the image of the near object is shifted to the retina and will be seen clearly.

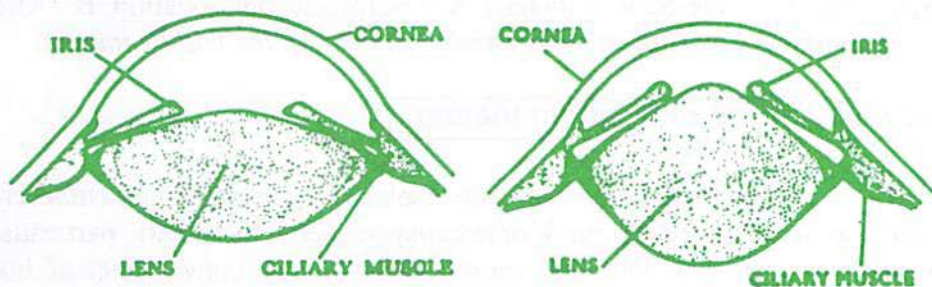


Figure 9 : Increased convexity of the anterior surface of the crystalline lens during accommodation (right). Compare with the resting eye (left).

During accommodation, *the increased convexity of the lens occurs mainly at its anterior surface* (figure 9). This is evidenced by studying the

PURKINJE-SANSON IMAGES : In a darkened room, a lighted candle placed in front of a subject (while looking far away) shows 3 images in *the subject's pupil* (figure 10 A). These are :

- (a) A *small upright image reflected from the cornea* (which acts as a convex mirror).
- (b) A *large upright image reflected from the anterior surface of the lens* (which acts as a convex mirror).
- (c) A *small inverted image reflected from the posterior surface of the lens* (which acts as a concave mirror).

The subject is *then asked to look at a near object* (figure 10 B). It will be noticed that the *middle image moves towards the first (corneal) image and becomes smaller* (indicating an increase in the curvature of the anterior surface of the lens), while the *first image shows no change* (indicating no change in the curvature of the cornea) and the *3rd image changes very little* (indicating insignificant change in the posterior surface of the lens).

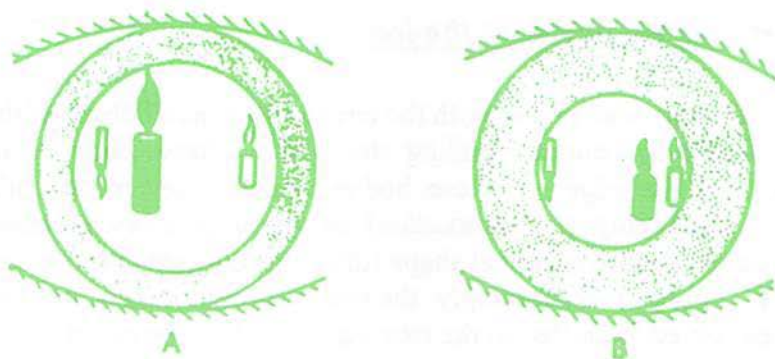


Figure 10 : Purkinje-Sanson images. A = Before accommodation. B = After accommodation (notice *miosis* and *movement of the middle image*).

Nervous control of accommodation

The 3 components of the near response are produced by the *oculomotor nerve* (so it is called the *nerve of accommodation*). Its somatic part causes contraction of the medial recti muscles (producing convergence of both eyes) while its parasympathetic part produces both pupilloconstriction and ciliary muscle contraction. Therefore, parasympatholytic drugs (e.g. atropine) paralyze accommodation (= *cycloplegia*) and also cause pupillo-dilatation (which increases both spherical and chromatic aberrations).

Nervous pathway of accommodation (figure 11)

The blurred retinal images initiate signals from the photoreceptors that are carried by the *optic nerve*. The *nasal fibres of this nerve cross* at the *optic chiasma* to the *contralateral optic tract* while the *temporal fibres proceed to the ipsilateral optic tract*, and all fibres then terminate at the *lateral geniculate body*. From the latter, signals are discharged and carried by new fibres called the *geniculocalcarine fibres*, which form the *optic radiation*. This radiation travels via the posterior limb of the internal capsule to the *visual cortex* (around the *calcarine fissure of the occipital lobe*) and its fibres relay in *area 17* (= visuo-sensory area). From the latter, signals arise and excite *area 18* (= visuo-psychic area) from which signals arise and excite *area 19* (= occipital eye-field area).

Signals from area 19 arise and are transmitted to the *superior colliculus of the midbrain* by the *occipitotectal tract* (via the anterior limb of the internal capsule) here they excite the *Edinger-Westphal nucleus* (= parasympathetic part of the oculomotor nerve nucleus). Preganglionic fibres arise at

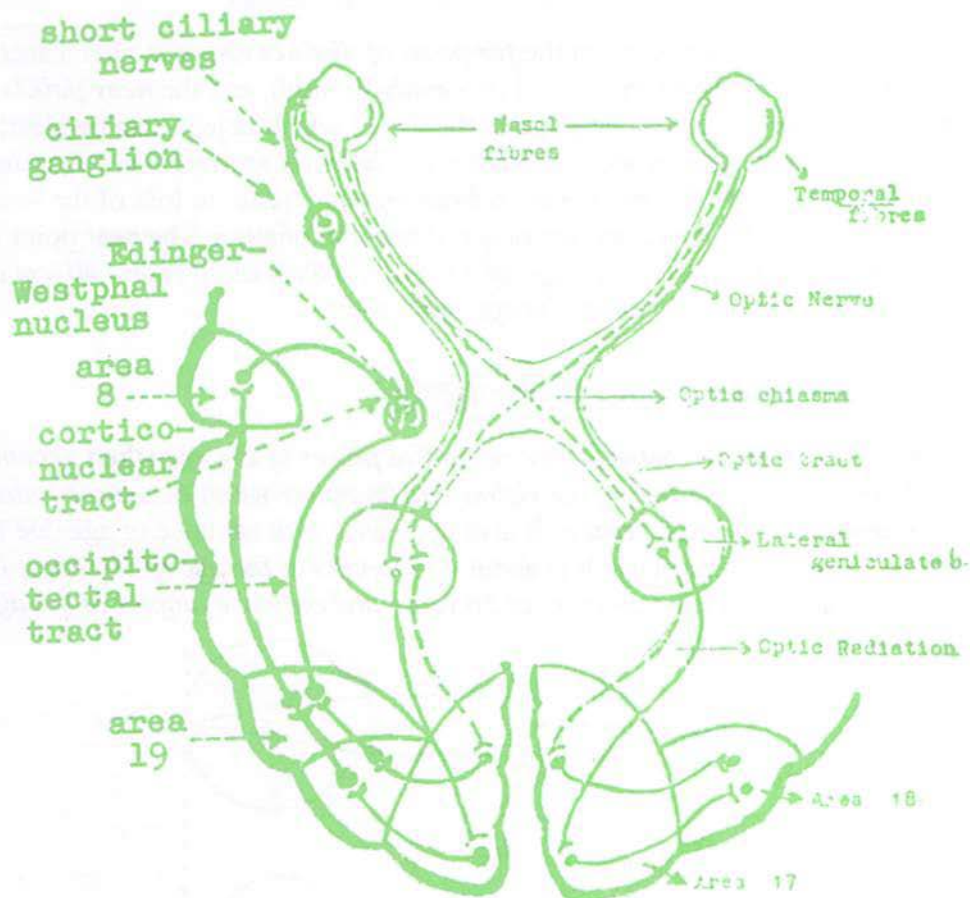


Figure 11 : Nervous pathway of accommodation.

this nucleus and relay at the *ciliary ganglion*, from which postganglionic fibres called *short ciliary nerves* arise and reach the eye (where they cause contraction of both the pupilloconstrictor as well as the ciliary muscles).

Stimulation of the somatic part of the oculomotor nerve nucleus (which results in contraction of the medial recti muscles) may also be produced by the occipitotectal tract. However, it is believed that signals are discharged from area 19 to the frontal lobe, where they stimulate *area 8 (= frontal eye-field area)*, from which a tract called the *cortico-nuclear tract* (figure 11) arises and descends via the anterior limb of the internal capsule to the midbrain, and the signals carried by this tract stimulate the somatic part of the oculomotor nerve nucleus.

Range of accommodation (the far & near points)

This is the distance between the **far point of distinct vision** at which accommodation is completely relaxed (*normally infinity*), and the **near point of distinct vision** (= the nearest point to the eye at which objects can be clearly seen through maximum accommodation). The latter *recedes* (i.e. becoming more distant from the eye) *with advancing age* (due to loss of the lens' elasticity), which decreases the range of accommodation. The near point is normally about 8.5 cm at the age of 15 years, 10 cm at 20 years, 40 cm at 50 years and almost 100 cm at the age of 60 years.

Amplitude (power) of accommodation

This is the *difference between the refractive power of the eye when accommodation is relaxed during far vision and its power when accommodation is maximal during near vision*. It also decreases with advance of age due to loss of the lens' elasticity. It is about 14 diopters at the age of 10 years, 10 diopters at 20 years, 2 diopters at 50 years and only one diopter at the age of 60 years.

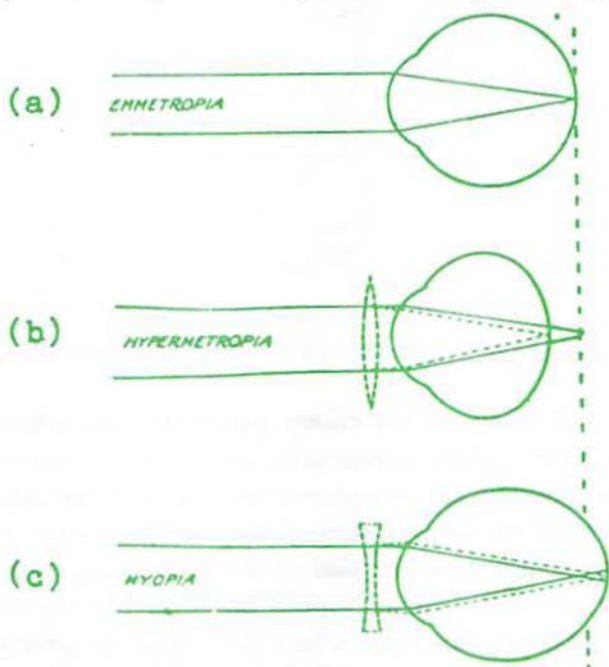


Figure 12 : (a) *Emmetropia* (parallel rays focus on the retina).
 (b) *Hypermetropia* (focus behind the retina and correction by a convex lens)
 (c) *Myopia* (focus in front of the retina and correction by a concave lens).

Presbyopia (= old age sight)

This is a *physiological decrease in the power of accommodation* that occurs in old age (starting at about **45 years**). It is due to gradual loss of the fluid state of the lens proteins (partly because of *progressive denaturation*) leading to *loss of its elasticity*. Accordingly, the power of accommodation is gradually decreased (becoming almost zero at the age of 70-75 years), and the *near point of distinct vision recedes far enough to make reading and other close works (e.g. threading a needle) difficult*. It is corrected by *convex lenses of suitable powers* (according to the severity of the condition).

Accommodation for distant vision may also be impaired (depending on the condition of the lens when it becomes stiff) and in this case, the patient *may also need correcting lenses for far vision*.

ERRORS OF REFRACTION

1. Hypermetropia (hyperopia, far sightedness or long sight)

This is an optical eye defect in which parallel rays striking the eye form a *focus behind the retina* (thus producing a blurred image). It is due to either (a) An abnormally short eyeball i.e. shortening of its anteroposterior diameter (figure 12 b) (b) An abnormally weak lens (less common).

The patient *continuously uses accommodation* (even during far vision) which leads to *eye fatigue and headache*. Also, persistent contraction of the medial recti muscles may produce convergent squint (specially in children).

The *far point in this case is normal*, while the *near point is much more further from the eye than normal*. Accordingly, patients with this defect can see distant objects but not the near ones (hence the name, long sight).

Correction: Hypermetropia is treated by *convex (+ve or converging) lenses* of a suitable power, depending on the degree of the defect (figure 12 b).

2. Myopia (short sightedness or short sight)

This is an optical eye defect in which parallel rays striking the eye form a *focus in front of the retina* (thus producing a blurred image). It is due to either (a) An abnormally elongated eyeball (figure 12 c) which becomes oval in shape (commonly as a result of stretching of a weak sclera in a rapidly-growing child by the normal intraocular pressure) (b) An abnormally greater curvature of the lens (or cornea) than normal, which increases its power.

The far point in this case is much limited, while the near point is much more close to the eyes than normal. Accordingly, patients with this defect can see near objects but not the distant ones (hence the name, short sight).

Correction : Myopia is treated by *concave (= - ve or diverging) lenses* of a suitable power depending on the degree of the defect (figure 12 c).



Figure 13 : The ciliary muscle in cases of emmetropia (a), hypermetropia (b), and myopia (c).

****** The range of accommodation is decreased in hypermetropia and myopia, while the amplitude of accommodation is increased in hypermetropia and decreased in myopia. On the other hand, the *ciliary muscle is well developed in hypermetropia and poorly developed in myopia* (figure 13), because it is continuously used in the former condition and almost not used in the latter.

3. Astigmatism

This is an optical error in which the *curvature of the cornea (or less commonly the lens) is not uniform*. It is often due to congenital causes but it may also be acquired e.g. following injury or inflammation (specially in the cornea). In some cases, there is an irregularity in the corneal (or lens) curvature (figure 14) but, however, *in most cases the cornea (or lens) is oblong or egg-shaped*. Such defects cause the curvature in one meridian (= plane) to be different from those in other meridians. Accordingly, light rays in that meridian are refracted to a different focus, leading to a blurred image (figure 15).



Figure 14 : Irregularities in the cornea and lens in case of astigmatism.

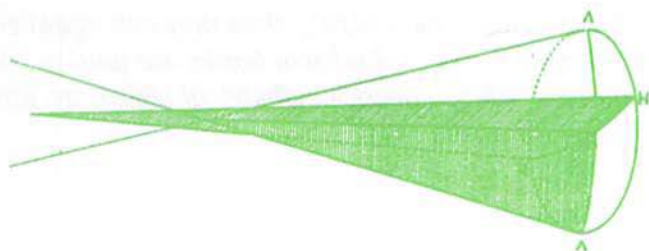


Figure 15 : Pathways of light rays in astigmatism. Notice that the vertical meridian (VV) is curved more than the horizontal meridian (HH).

Astigmatism commonly affects the 2 chief meridians of the cornea i.e. the vertical and horizontal meridians (but it may also affect other meridians). *In most cases, the cornea has maximum curvature in the vertical meridian and least curvature in the horizontal meridian.* This is called **astigmatism with the rule** (figure 15). When reverse conditions are present, it will be against the rule. *Normally, there is a slight degree of astigmatism with the rule.*

Types of astigmatism

There are 2 main types of astigmatism : **regular and irregular**. In both types, there is a difference in the degree of refraction in the 2 principal meridians. However, *in the regular type, the refraction in each meridian is the same throughout*, while in the irregular type, the refraction at the different parts of the same meridian is unequal. **Regular astigmatism is further subdivided into the following types :**

(1) **Simple astigmatism** : In this case, one meridian is emmetropic (i.e. normally formed) while the other is either myopic or hypermetropic. These types are called *myopic and hypermetropic astigmatism* respectively.

(2) **Compound astigmatism** : In this case, both meridians are myopic or hypermetropic, but in either case, the degree of the defect is *unequal in both meridians*.

(3) **Mixed astigmatism** : In this case, one meridian is myopic while the other is hypermetropic.

Correction of astigmatism

Astigmatism is corrected by **cylindrical lenses** (portions of a cylinder cut longitudinally). These lenses **correct the error of refraction in one meridian**

only (without affecting other meridians), *thus they can equalize the refraction in all meridians*. Convex cylindrical lenses are used to correct hypermetropic astigmatism, while concave cylindrical lenses are used to correct myopic astigmatism.

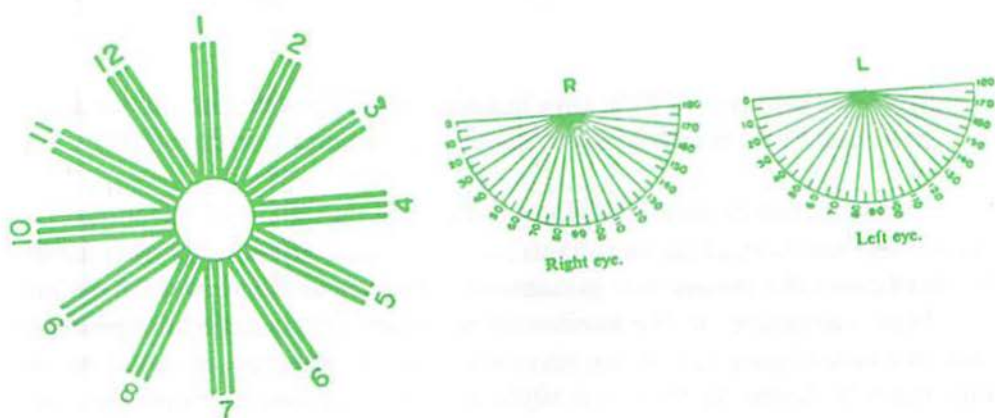


Figure 16 : 2 types of charts for determination of the axis of astigmatism.

The strength of the lens is determined according to the extent of the underlying irregularity while the affected meridian (i.e. *the axis or plane of astigmatism*) is determined by special *astigmatic charts* (figure 16), and the curvature of the correcting lens should be placed in the same plane of the affected meridian. In cases of severe corneal irregularities, *contact lenses* can be used (which provide a smooth uniform surface).

CHAPTER 4

THE MIDDLE LAYER OF THE EYE (THE UVEAL TRACT)

The middle layer of the eye is rich in blood vessels and it consists of the choroid posteriorly, the iris anteriorly and the ciliary body in between (figure 1).

Functions of the choroid

- (1) It is a *vascular layer* which provides blood supply to the eye.
- (2) The pressure inside its vessels *maintains the intraocular pressure*.
- (3) It is rich in *melanin*, which forms a dark coating for the interior of the eyeball. This is important because it prevents reflection of light rays inside the eye (which increases its optical efficiency).
- (4) It gives *attachment to the ciliary muscle*.

Functions of the ciliary body

This contains the *ciliary muscle and the ciliary processes*.

- (1) The ciliary muscle is essential for *accommodation* (page 13).
- (2) The ciliary processes secrete the *aqueous humour* into the posterior chamber and also give attachment to the suspensory ligament of the lens.

THE IRIS

This is a circular diaphragm-like opaque structure that lies anterior to the crystalline lens. Its margins rest on the anterior surface of the lens, and in front of it there is the *anterior chamber* while behind it there is the *posterior chamber* (figure 1). It has a central round aperture (opening) called the *pupil*, the diameter of which is greatly variable (it may be as small as 1.5 mm or as large as 8 mm).

The iris contains the pigment that gives the eye its *characteristic colour* (particularly in its posterior layer). It also contains the following 2 types of smooth muscle which *control the size of the pupil* :

- (1) Circular muscle fibres, which constitute the *constrictor (or sphincter) pupillae muscle*.
- (2) Radial muscle fibres, which constitute the *dilator pupillae muscle*.

Functions of the iris

- (1) It *regulates the entry of light rays* into the eye through the pupil.
- (2) It *prevents both spherical and chromatic aberrations* by cutting out most of the peripheral rays falling on the eye (page 12).
- (3) It *protects the retina* (by preventing excessive entry of ultraviolet rays into the eye).
- (4) The *pupillary reflexes* are important in the diagnosis of many nervous lesions and diseases (see later).

Nerve supply of the iris muscles

The constrictor pupillae muscle receives parasympathetic fibres while the dilator pupillae muscle receives sympathetic fibres.

(A) Parasympathetic nerve supply (pathway of pupilloconstriction)

The centre of pupilloconstriction is the *Edinger-Westphal nucleus* (= parasympathetic part of the oculomotor nerve nucleus in the midbrain). Pregang. fibres arise from this nucleus and run in the oculomotor (= 3rd cranial) nerve. These fibres relay in the *ciliary ganglion*, from which postgang. fibres called the *short ciliary nerves* arise and enter the eye where they supply the constrictor pupillae muscle leading to pupilloconstriction.

Effects of stimulation of the ocular parasympathetic nerves

- (1) Stimulation of the *oculomotor nerve* produces (a) Pupilloconstriction (= *miosis*) (b) Contraction of the *ciliary muscle* (page 14).
- (2) Stimulation of the *facial nerve* produces *profuse lacrimation*.

Effects of oculomotor nerve injury

- (1) *Squint* (due to paralysis of certain external ocular muscles).
- (2) *Pupillodilatation* (due to predomination of sympathetic activity), which may cause *spherical and chromatic aberrations* (page 12).
- (3) Paralysis of accommodation (= *cycloplegia*), which leads to difficulty of reading as well as inability to perform close works.

****** Effects 2 and 3 are also produced by *atropine drops* (which blocks the action of the parasympathetic chemical transmitter i.e. acetylcholine).

(B) Sympathetic nerve supply (pathway of pupillodilatation)

The centre of pupillodilatation is located in the *lateral horn cells of the upper 2 thoracic segments (= ciliospinal centre)*. Pregang. fibres arise from this centre, ascend in the sympathetic chain and relay at the *superior cervical ganglion*. Postgang. fibres arise from this ganglion, run along branches of the internal carotid artery and enter the eye as the *long ciliary nerves* where they supply the dilator pupillae muscle leading to pupillodilatation

The ciliospinal centre is controlled by signals discharged from the *hypothalamus* (which descend to the spinal cord via the reticulospinal tract)

Effects of stimulation of the ocular sympathetic nerves

- (1) Pupillodilatation (= *mydriasis*).
- (2) Retraction of the upper eyelid (resulting in *widening of the palpebral fissure*) due to contraction of the involuntary smooth muscle (= superior tarsal muscle) in the deep part of the levator palpebrae superioris muscle.
- (3) *Exophthalmos* (specially in animals) due to contraction of Muller's muscle (which is located behind the eyeball).
- (4) *V.C. of the blood vessels of the eye.*
- (5) *A small amount of concentrated lacrimal secretion.*

Effects of ocular sympathetic nerve injury (= Horner's syndrome)

All the following effects occur in the same side of the injury :

- (1) *Miosis* (= pupilloconstriction).
- (2) *Ptosis* (= drooping of the upper eyelid).
- (3) *Enophthalmos* (= sinking of the eyeball backwards).
- (4) *Anhydrosis* (= loss of sweat secretion).
- (5) *V.D. in the face* (so it appears more red than the other side).

The pupillary light reflex

When one eye is exposed to light, pupilloconstriction occurs in this eye (= *direct reflex*) as well as in the other eye (= *consensual reflex*). The response in the stimulated eye *protects its retina from excess light*. Testing for this reflex is important clinically in (a) Diagnosis of certain nervous diseases (b) Localization of the sites of lesions in the visual pathway (c) Determination of the depth of anaesthesia (see later).

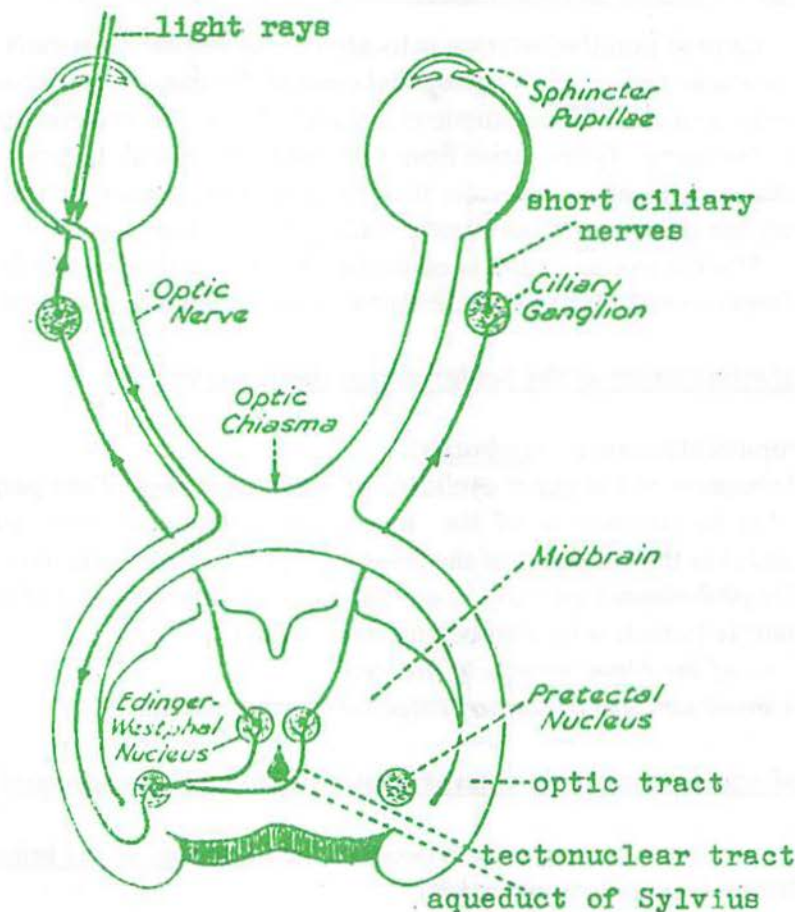


Figure 17 : Nervous pathway of the pupillary light reflex.

Nervous pathway of the pupillary light reflex

The photoreceptors of the stimulated retina generate signals that are transmitted by afferent fibres in the **optic nerve to the optic chiasma** then to the **optic tract** (of the opposite side if light rays were falling on the nasal side of the retina, and of the same side if light rays were falling on the temporal side of the retina as in figure 17).

Collateral fibres leave the optic tract and terminate at the **pretectal nucleus in the midbrain (centre of the reflex)**. Fibres then arise from the pretectal nucleus (= **tectonuclear tract**) and terminate at the **Edinger**

Westphal nuclei of both sides (the fibres that reach the opposite side cross close to the *aqueduct of Sylvius*). Pregang. fibres arise from the Edinger Westphal nuclei and relay in the *ciliary ganglia*, from which postgang. fibres (*short ciliary nerves*) arise and enter the eyes, where they lead to contraction of the constrictor pupillae muscles, resulting in *bilateral miosis*.

The Argyll Robertson pupil

This is a *pupil that does not constrict in response to light but constricts during accommodation*. It is a pathological condition that occurs in diseases producing selective lesions in the midbrain, most commonly *neurosyphilis*. This disease causes *damage of the pretectal region*, thus the light reflex is lost (because its pathway is interrupted) while the accommodation reflex remains intact, because its pathway is not affected (page 14).

Conditions that cause pupilloconstriction (miosis)

- (1) **Reflexes** : The commonest reflexes associated with miosis are the pupillary light reflex and the near (accommodation) reflex (see above).
- (2) **Interruption of the ocular sympathetic nerve supply** : This occurs in
 - a. Injury of the ciliospinal centre and Horner's syndrome (see above).
 - b. Spinal cord transection in the cervical region and pontine haemorrhage (due to interruption of the hypothalamic pupillo-dilating signals)
- (3) **During sleep** : Miosis occurs during sleep due to (a) Predominance of parasympathetic activity (b) Release of the Edinger Westphal nucleus from the normal cortical inhibition (because the cortex itself is depressed).
- (4) **Sudden fall of the intraocular pressure** e.g. after sudden opening of the anterior chamber (due to passive V.D. of the iris vessels).
- (5) **During rotation** (due to stimulation of the semicircular canals).
- (6) **During the 3rd stage (= surgical stage) of anaesthesia** (see below).
- (7) **Prolonged irritation of the trigeminal nerve** (due to reflex V.D. of the iris vessels)
- (8) **Miotic drugs** : These include the following :
 1. **Parasympathomimetic drugs** : These act either :
 - a. *Directly* (by stimulating the muscarinic receptors in the constrictor pupillae muscle) e.g. *pilocarpine*.
 - b. *Indirectly* (by inhibiting the cholinesterase enzyme) e.g. *eserine* (= physostigmine).
 2. **Morphine** : This depresses the cerebral cortex, so the Edinger Westphal nucleus is released from its inhibitory effect, resulting in miosis.

Conditions that cause pupillodilatation (mydriasis)

- (1) Conditions of *sympathetic stimulation & excess secretion of adrenaline* e.g. (a) Emotions (fear, anger, etc.) (b) Pain (c) Asphyxia (d) Stress.
- (2) *Stimulation of the ocular sympathetic nerve supply* through the *cilio-spinal reflex* (pinching the skin of the neck initiates signals that stimulate the cilio-spinal centre, leading to pupillodilatation).
- (3) *During distant vision* (i.e. when accommodation is relaxed).
- (4) *Sudden withdrawal of light from the eyes.*
- (5) *During dark adaptation* (see next chapter).
- (6) *Injury of the ocular parasympathetic nerve supply* e.g. due to lesions of the oculomotor nerve or the Edinger Westphal nucleus.
- (7) *Following sudden stoppage of rotation.*
- (8) *An increase of the intraocular pressure* e.g. in glaucoma (due to passive V.C. of the iris vessels).
- (9) *During brain concussion* (after a head injury).
- (10) *During the induction and postsurgical stages of anaesthesia.*
- (11) *Short-term irritation of the trigeminal nerve.*
- (12) *Alcohol intoxication and chloroform poisoning.*
- (13) *Hyperthyroidism* (because thyroxine sensitizes the dilator pupillae muscle to the action of epinephrine).
- (14) ***Mydriatic drugs*** : These include the following :
 1. *Parasympatholytic drugs* e.g. atropine and homatropine (the latter acts for a shorter time than atropine), which block the muscarinic receptors in the constrictor pupillae muscle.
 2. *Sympathomimetic drugs* (e.g. epinephrine), which stimulate the alpha adrenergic receptors in the dilator pupillae muscle.
 3. *Cocaine* : This sensitizes the dilator pupillae muscle to the action of epinephrine and sympathetic impulses (so cocaine eye drops produce mydriasis in addition to corneal anaesthesia).

Effect of anaesthesia on the size of the pupil

Anaesthesia passes in 4 stages, during which the size of the pupils varies as follows :

- (1) *In the first stage (= first induction stage)*, the pupils have normal size (or they are slightly dilated).
- (2) *In the second stage (= second induction stage)*, the pupils are widely dilated due to *epinephrine secretion from the adrenal medullae and increased sympathetic activity* (as a result of emotional excitement).

- (3) *In the third stage (= surgical stage), the pupils are constricted due to release of the Edinger Westphal nucleus from the normal cortical inhibition (because the cerebral cortex itself is depressed). This stage is the proper stage to start surgical procedures.*
- (4) *In the fourth stage (= postsurgical stage), the pupils are dilated due to paralysis of the Edinger Westphal nucleus. It is an indication to stop anaesthesia, because administration of more anaesthesia would depress the lower nerve centres in the medulla oblongata, which is dangerous (and may be fatal) due to depression of the respiratory centres.*

N.B.

During anaesthesia, pupil dilatation occurs in both the induction stages as well as the postsurgical stage, and it is essential to differentiate between these stages because anaesthesia is indicated in the induction stages but it is dangerous in the postsurgical stage (see above). Such differentiation is achieved by examining (a) The corneal reflex (b) The pupillary light reflex (c) The movements of the eyes. These are intact in the induction stages but lost in the postsurgical stage.

CHAPTER 5

THE RETINA

The retina is a *nervous structure* forming the inner layer of the eye. It constitutes the light-sensitive part of the eye, since it contains the visual receptors (or photoreceptors) called the *rods and cones*.

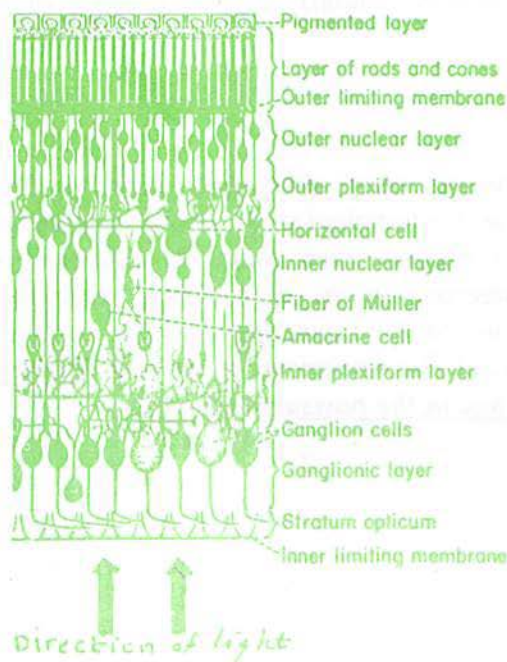


Figure 18 : Structure of the retina.

Structure of the retina

Histologically, the retina is formed of 10 layers (figure 18), the most important of which include the following :

(1) *The layer of rods and cones* : This layer lies next to the pigmented layer (which merges into the choroid).

(2) *The outer nuclear layer* : This includes the inner segments of the rods and cones, which constitute the *first order neurons in the visual pathway* (see below).

(3) **The inner nuclear layer (the layer of bipolar cells):** This constitutes the *second order neurons in the visual pathway*.

(4) **The layer of ganglion cells :** These cells give rise to the optic nerve fibres (which constitute the *third order neurons in the visual pathway*).

The retina also contains 2 types of cells called the **horizontal and amacrine cells** (figure 28), which exert characteristic functions (see below).

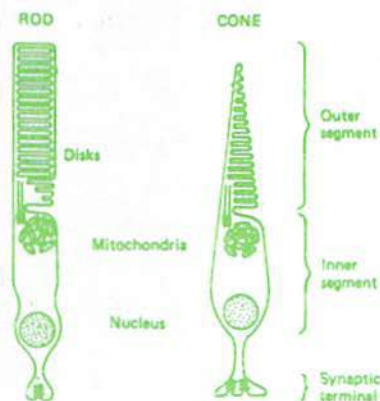


Figure 19 : Structure of the rods and cones.

Structure of the rods and cones

Each rod or cone consists of **an outer and an inner segment** (the latter forms the outer nuclear layer and is rich in mitochondria) as well as a **synaptic body or terminal** (figure 19). The outer segments are modified cilia, and are **thin rod-like in rods but conical in cones**. They are made up of **regular flattened saccules or discs** (about 1000 in each rod or cone). In **cones**, each disc is actually an infolded shelf of cell membrane but in **rods** the discs separate from the membrane and form flat sacs lying totally inside the cell. These discs and sacs contain the **photosensitive compounds** that react to light. Such compounds are so concentrated that they constitute 40-90 % of the mass of the outer segments. The old (outer) discs are continuously phagocytized (by cells of the pigment epithelium) and are constantly renewed (by formation of new discs at the inner edges of the segments).

The **rods have a uniform shape** all over the retina while the cones vary in shape (they are **long & thin in the fovea and shorter & broader in the periphery of the retina**). The inner processes of the rods & cones synapse with the dendrites of the bipolar cells, the axons of which synapse in turn with the dendrites of the ganglion cells. The axons of the ganglion cells collect and form the optic nerve fibres which leave the eye at **the optic disc** (figure 20).

****** Since the layer of rods and cones is the outermost layer in the retina (lying next to the choroid), **light rays have to pass first through the various layers of the retina before reaching these photoreceptors** (figure 18) which would decrease the visual acuity. However, *this minimally occurs in the fovea centralis* (see below).

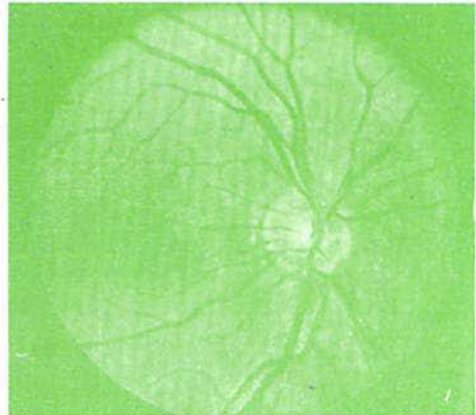
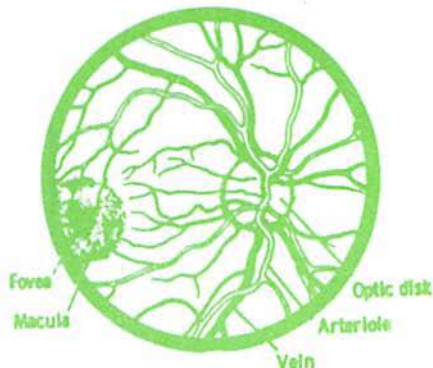


Figure 20 : The normal retina as seen through the ophthalmoscope (right) and an illustrating diagram (left).

Blood supply of the retina

The layer of rods and cones is supplied by the **choroidal capillaries** while all the remaining retinal layers are supplied by the **central retinal artery** (a branch from the ophthalmic artery that enters the eye through the optic disc *among the optic nerve fibres*).

Advantage and disadvantage :

The retinal system of blood supply is a **disadvantage in cases of occlusion of the central retinal artery** because this artery supplies most layers of the retina and it makes *no anastomoses with the choroidal blood vessels* (so its occlusion leads to sudden and complete blindness).

However, the retinal system of blood supply is an **advantage in cases of retinal detachment**. This is because the independent blood supply of the retina (and also the diffusion of fluid across the detachment gap) renders the retina in such cases able to resist degeneration for several days (so the normal function of the retina can be resumed if it is rapidly replaced into its normal position).

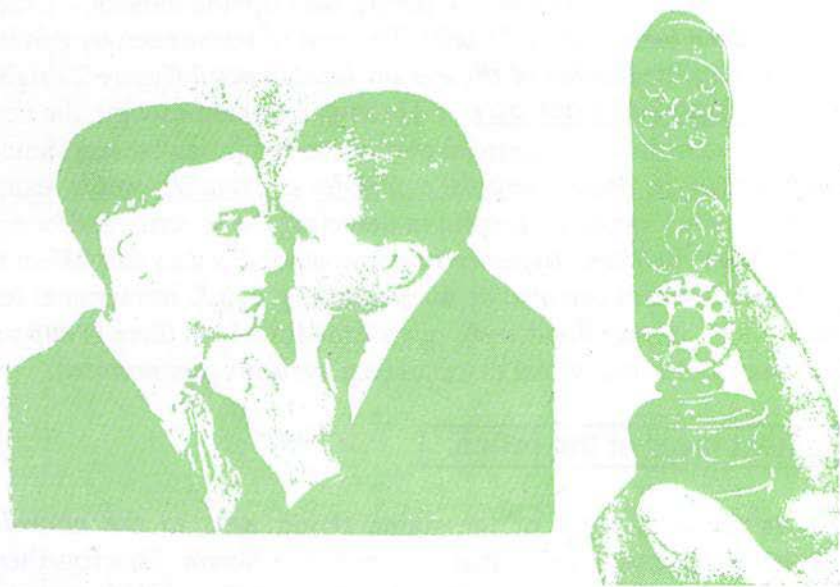


Figure 21 : The ophthalmoscope (right) & the method of examination (left).

The ophthalmoscope

This is an instrument used for examination of the internal structures of the eye particularly the retina (figure 21 right).

Principle of ophthalmoscopy : A part of a light beam entering the eye is reflected back from the retina in its original course. If the observer and the source of light are in the same position, the reflected rays from a patient's eye can be perceived by the observer. In ophthalmoscopy, light is reflected from a plane or concave mirror into the patient's eye and the observer's eye is placed just behind a hole in the mirror (thus he can see the *fundus glow*). In figure (22), if the observer's eye is in position (E), he will see only point (B) of the patient's retina, but if the observer comes closer to the patient and assumes position (E₁), he will see points (A) and (C) as well.

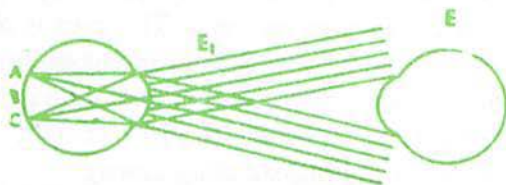


Figure 22 : Principle of ophthalmoscopy.

Procedure of ophthalmoscopy : A few drops of homatropine are put into the patient's eye (to dilate the pupil), then ophthalmoscopic examination is carried out (as in figure 21 left). The part of retina seen by this instrument is known as the *fundus of the eye or fundus oculi* (figure 20 right).

Benefits of ophthalmoscopy : Through ophthalmoscopy, the arteries, arterioles and veins in the superficial part of the retina can be seen. Since this is the *only site in the body where the arterioles are readily visible*, examination of the fundus oculi is helpful in detecting the severity and complications of *diabetes mellitus*, *hypertension* and other diseases that affect blood vessels. Other diseases can also be diagnosed e.g. a high intracranial tension (in which there is *papilloedema*), glaucoma (in which there is *cupping of the optic disc*) and some retinal diseases (e.g. *retinitis pigmentosa*).

The pigment layer of the retina

This is the first layer of the retina (lying *next to the choroid*). It contains large epithelial cells that are rich in *melanin*. This together with the pigment in the choroid act like the *black paint inside a camera*. It *absorbs the light rays that are not absorbed by the photoreceptors*, thus preventing their reflection back on the retina, which would lead to blurring of vision as a result of stimulation of a large number of photoreceptors at the same time (as occurs in albinos). Therefore, the pigment layer is essential for *acute vision*, and in addition, it *stores considerable amounts of vitamin A*, which plays an important role in retinal function (see later).

The optic disc (= optic papilla or blind spot)

This is the site where the optic nerve fibres leave the eyeball and the central retinal artery enters it (figure 20). It is a *pinkish white circular* (or oval) area having *a diameter about 1.5 mm*, and is located about *3 mm medial to and slightly above the posterior pole of the eye*. It contains nerve fibres only but *no photoreceptors* (so it cannot perceive light and, accordingly, it is a *physiological blind spot*).

At the optic disc, the optic nerve leaves the eyeball through an area in the sclera known as the *lamina cribrosa*. This area is *the weakest part in the eyeball*, so if the intraocular pressure increases (e.g. in glaucoma), it bulges out (= *cupping of the optic disc*) and the optic nerve fibres are compressed against its rim (which would lead to atrophy of the optic nerve fibres and may be blindness in long-standing cases).

THE RODS AND CONES

These are the *photoreceptors of the retina*. They contain specific *photosensitive pigments* which are chemically changed on exposure to light, and such changes initiate signals (action potentials) in the optic nerve fibres.

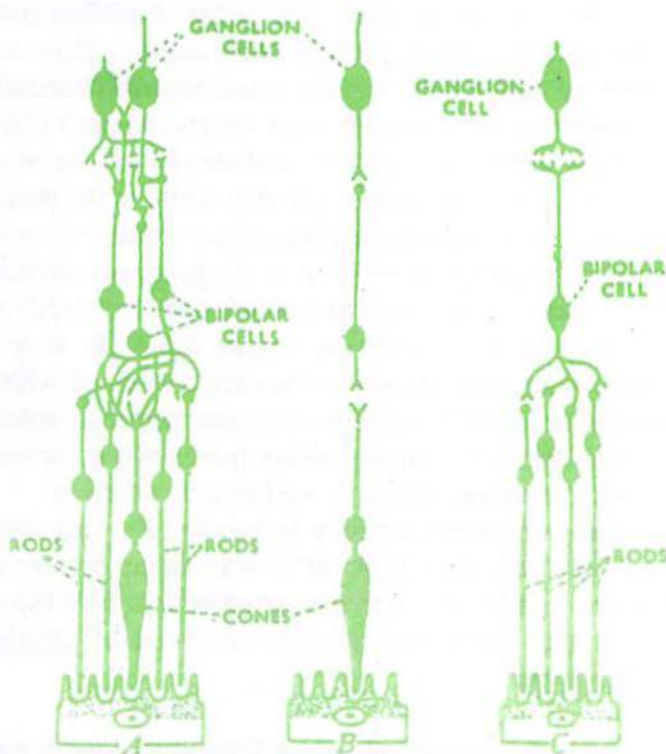


Figure 23 : Distribution and types of connections of the photoreceptors
(A) peripheral to the fovea, (B) in the fovea (cones only),
(C) in the most peripheral parts of retina (rods only).

THE RODS

In each human retina, there are about **120 million rods** which are present *mostly at the periphery of the retina*. They are few in the central part of the retina and *absent in the fovea centralis*. Their photochemical pigment is called *rhodopsin (or visual purple)*, which is extremely sensitive to *dim (= faint) light*.

Therefore, the *peripheral vision* (perceived by rods) is responsible for *twilight or night vision (= scotopic vision)* in which the details of objects

and their colours cannot be perceived. As much as 100-200 rods converge on a single optic nerve fibre (figure 23), and such arrangement allows for *summation of impulses from the rods*.

THE CONES

In each human retina, there are about **6 million cones**, which are present *mostly at the central part of the retina*. They are few in the peripheral parts of the retina, and the *fovea centralis contains only cones* (figure 23). There are **3 types of cones** which contain 3 different types of photochemical pigments responsible for *colour vision* (see later).

In the fovea, the cones are long & thin while in the periphery they are shorter & broad. In the peripheral parts of the retina, cones and rods often converge on the same ganglion cell, but *in the fovea no convergence occurs* and *each foveal cone is connected to a single optic nerve fibre* (figure 23).

Cones are stimulated only by **bright light**, so they are *much less sensitive to light than rods*. However, they are concerned with *day vision* (= *photopic vision*) in which the details of objects and their colours are clearly perceived. Therefore, the *central vision* (perceived by cones in the fovea) allows for *acute (or sharp) vision as well as colour vision*.

Since cones are stimulated only by bright light, they *are not acting at night*. Accordingly, the dim light of a star cannot be seen if fixed on the fovea but becomes visible if vision is everted peripherally (so as to bring the star's image on the rods). This phenomenon is called **physiological night blindness of the fovea**.

The following table summarizes the *differences between rods and cones*.

	Rods	Cones
Number	120 million / retina	6 million / retina
Distribution	mostly peripheral	mostly central
Photosensitive pigment	Rhodopsin	3 different types
Light sensitivity	Up to 300 times as cones	Low
Threshold of stimulation	Low	High
Convergence	Well marked	Very little (absent in the fovea)
Function	Scotopic (night) vision	Photopic (day) vision and colour vision
Acuity of vision	Low	Very high
Shape	Uniform allover	Variable (see text)

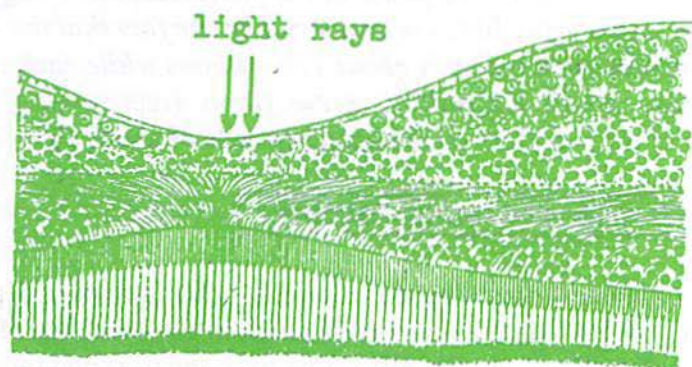


Figure 24 : Structure of the retina at the fovea centralis (notice its thinning).

The macula lutea and fovea centralis

These are the most important parts of the retina because they are specialized for *perception of the details of objects and their colours*. The macula lutea (= yellow spot) is *less than 1 mm²* and is located about *3 mm on the temporal (lateral) side of the optic disc* (i.e. at the centre of the retina opposite to the posterior pole of the eye). It contains *mainly cones* (but few rods are also present).

The fovea centralis is a small thin area *at the centre of the macula (about 0.4 mm in diameter)*. It is highly developed in man and contains *only densely-packed cones* (about 35000). Its structure allows *maximal degree of distinct vision* because :

- (1) It contains *no blood vessels* (which increases the visual acuity).
- (2) In the fovea, the *retina is thinned out* due to compression of its layers, so some of these layers are displaced to the sides (figure 24). Such arrangement *allows the light rays to reach the cones directly* without scattering at various cellular layers (which also increases the visual acuity).
- (3) Each foveal cone is connected to a single optic nerve fibre, i.e. has a private pathway to the cerebral cortex (figure 23), which also allows maximal visual acuity.
- (4) The pigmented layer of the retina is highly developed in the foveal region, thus light reflection at this region is greatly prevented (which also allows a high degree of acute vision).

****** Because the fovea is the area of maximal visual acuity, on looking to an object, the eyes normally move so that light rays coming from that object are focused on the fovea.

****** The phenomenon of convergence of the photoreceptors (particularly the rods) on the optic nerve fibres is *evidenced by the fact that the number of these receptors in each retina is about 126 millions while each optic nerve contains only about 1.2 million nerve fibres* (representing an overall convergence of the receptors on the ganglion cells of about 105 : 1).

The duplicity theory of retinal function

This theory states that retinal function is *double in nature* depending on the properties of its 2 photoreceptors. It assumes the existence of 2 kinds of inputs to the C.N.S. from the retina (one from the rods and the other from the cones) each of which works maximally under a different condition of illumination. *Rods have a very low threshold of stimulation* (i.e. they are *extremely sensitive to light*), thus they are the receptors of *twilight or night vision* (i.e. *scotopic vision*) in which the details of objects and their colours are not accurately perceived. On the other hand, *cones have a much higher threshold of stimulation than rods* (i.e. *they are much less sensitive to light*), thus they are the receptors of *day vision* (i.e. *photopic vision*) in which the visual acuity is maximal (i.e. the details of objects and their colours are accurately perceived).

Evidences of the duplicity theory

(1) The feeble (faint) light of a star cannot be seen if fixed on the fovea which contains cones only (= *physiological night blindness of the fovea*).

(2) Animals and birds adapted for night-life (e.g. owls and rats) have rods only in their retinae, while those adapted for day-life (e.g. chicken) have cones only.

(3) Determination of the visual fields of different colours showed that the peripheral parts of the retina (which contain mainly rods) are colour blind while the central part is most sensitive to colours.

(4) Vitamin A deficiency, which causes dysfunction of the rods (due to deficient formation of rhodopsin), leads to *night blindness* (= *nyctalopia*) but does not affect day vision.

(5) Dog's retinae contain rods only, and they were proved by Pavlov to be colour blind (so they couldn't develop conditioned reflexes based on differentiation of various colours).

(6) The eye shows both dark and light adaptation (see below). This suggests the existence of 2 different visual mechanisms in the retina.

Objections to the duplicity theory

(1) Guinea pigs have rods only in their retinae, but they can see during day time and can also differentiate some colours.

(2) Snakes have cones only in their retinae, but they can see at night and cannot differentiate various colours.

Modification of the duplicity theory

Cones are the main receptors of photopic vision, but they also share in scotopic vision (e.g. in snakes). *Rods, on the other hand, are the main receptors of scotopic vision*, but they also share in photopic vision (e.g. in guinea pigs) and can perceive some colours (particularly the blue colour).

THE VISIBLE SPECTRUM

The wavelengths of visible spectral colours range from **400 to 725 nm (nanometer or millimicron)**, and beyond this range, the retinal photoreceptors are not stimulated, thus ultraviolet rays (below 400 nm) and infrared rays (above 725 nm) are normally invisible. The wavelengths of various colours show some overlap, but however each is maximum at a certain wavelength as follows : **violet (400 nm), blue (445 nm), green (535 nm), yellow (565 nm), orange (600 nm) and red (650-725 nm)**.

The spectral zone that appears more luminous differs according to the intensity of illumination as shown in the ***scotopic and photopic visibility (or luminosity) curves*** (figure 25). These curves show the magnitude of retinal sensitivity (and in turn, the degree of luminosity) during photopic and scotopic vision (the maximal sensitivity is taken as 1 in both cases). During photopic vision the maximal retinal sensitivity (i.e. maximal luminosity) is obtained at a wavelength of 550 nm i.e. at the **yellow-green zone** of the spectrum whereas during scotopic vision, maximal luminosity is obtained at a wavelength of 505 nm i.e. at the **blue-green zone** of the spectrum.

Purkinje shift (or phenomenon)

This is the ***shift of spectral sensitivity from the yellow-green zone in day light to the blue-green zone in dim light***. Such phenomenon explains why red and blue flowers look almost equally bright during day-time, but as light fades, the blue flowers are still seen clearly while the red flowers appear black.

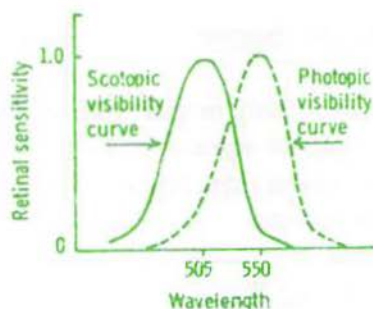


Figure 25 : The scotopic and photopic visibility (or luminosity) curves.

Spectral absorption curves of photoreceptor pigments

These curves are constructed *in vitro*, and they determine the absorption of various wavelengths of light by solutions of the photoreceptor pigments. It was found that solutions of rhodopsin (the rod pigment) absorb the spectrum maximally at a wavelength of 505 nm, similar to the scotopic visibility curve (figure 26). On the other hand, solutions of the cone pigments were found to absorb the spectrum maximally at a wavelength of 550 nm (similar to the photopic visibility curve). The similarity between the visibility curves and the spectral absorption curves of photoreceptor pigments proves that rods are the receptors of scotopic vision while cones are the receptors of photopic vision (*which is an additional evidence to the duplicity theory*).

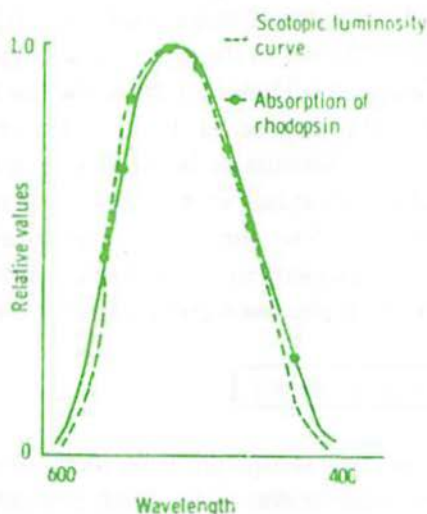


Figure 26 : Similarity between the scotopic luminosity curve and the spectral absorption curve of rhodopsin.

NEURAL FUNCTION OF THE RETINA

Mechanism of signal transmission in the retina

The electric responses in the neural elements of the retina are in the form of *local potentials*, and the conduction of visual signals in all retinal cells occurs by *electrotonic conduction* (i.e. direct flow of electric current in the neuronal cytoplasm from the point of excitation to the output synapses) *except in the ganglion cells (in which conduction occurs by all or none action potentials)*. Such property of electrotonic conduction allows *graded conduction of signal strength*.

Photoreceptor potentials

In the dark, the inner segments of the retinal photoreceptors (rods and cones) continuously pump Na^+ outwards (by activity of $\text{Na}^+ - \text{K}^+$ ATPase) and the membranes of their *outer segments are very leaky to Na^+* (figure 27) *by effect of cyclic GMP*. Therefore, Na^+ continually re-enters these receptors (via their outer segments), leading to decreased negativity inside the receptors, and the potential is maintained at about -40 mV.

On the other hand, on exposure to light, the retinal receptors are excited and a receptor potential develops. However, the developed potential is *normally hyperpolarizing* (and not depolarizing as in most other sensory receptors). This occurs through *a cascade of reactions* as follows :

1. When light strikes the outer segments of the receptors, it is absorbed by the photosensitive pigments leading to their *activation* (i.e. a change of their structures).
2. The activation of the photosensitive pigments leads to formation of a certain substance (unknown in the cones, and metarhodopsin II in rods) which activates a G protein (called *transducin* in rods and a similar protein in cones).
3. The activated G proteins, in turn, activate the phosphodiesterase enzyme, which catalyzes breakdown of cyclic GMP. Accordingly, the intracellular content of cyclic GMP is decreased
4. The decreased cyclic GMP leads to closure of the Na^+ channels in the outer segments of the receptors, while Na^+ is continuously pumped out from their inner segments. This creates marked negativity inside the receptors and marked positivity outside them, leading to a state of hyperpolarization, and the potential becomes -70 to -80 mV at maximal light intensity.

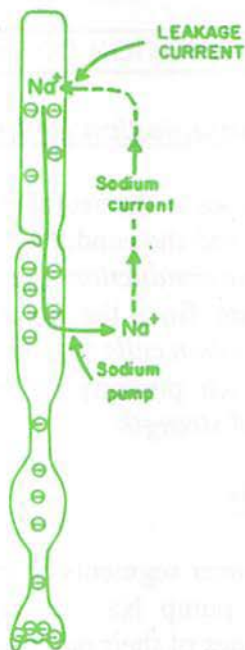


Figure 27 : current flow in the photoreceptors in the dark.

****** Such cascade of reactions occurs very rapidly and also *amplifies the light signal*. This amplification explains the high sensitivity of the photoreceptor pigments *specially rhodopsin* (which is capable of producing a detectable response to as little as one photon of light).

****** The release of transmitter from the synaptic terminals of the photoreceptors (which is probably *glutamate*) is *steady in the dark*, but on exposure to light, *the resulting hyperpolarization reduces the release of the synaptic transmitter* and this generates a signal (in an unknown way) that leads to action potentials in the ganglion cells.

****** when a sudden pulse of light strikes the retina, the state of hyperpolarization of the photoreceptors reaches a peak in about 0.3 second and lasts for a little more than one second then disappears (so the Na^+ channels reopen and the resting potential is restored). This is produced as a result of :

(a) Inactivation of the substance that activated the G protein by certain enzymes (e.g. metarhodopsin II in the rods is inactivated by an enzyme called *rhodopsin kinase*).

(b) Resynthesis of cyclic GMP. This occurs as follows : Light decreases the concentration of Ca^{++} in the photoreceptors (together with decreasing their Na^+ content) and this *activates the guanylyl cyclase enzyme (and also inhibits the phosphodiesterase enzyme)*, which increases the intracellular content of cyclic GMP.

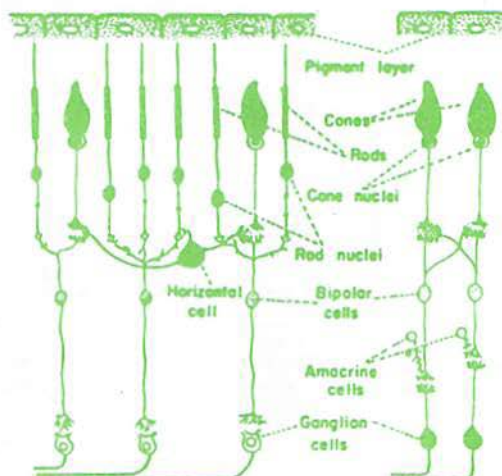


Figure 28 : Neural organization of the retina at the fovea (right) and at the peripheral parts (left).

Electrical responses and functions of the retinal cells

The responses of the various retinal cells are produced by a large number of neurotransmitters that are released in the retina (see below), and they respond by either *depolarization or hyperpolarization*. Such responses as well as the functions of each cell are as follows :

(1) **The rods and cones** : The response of these photoreceptors is *hyperpolarizing* (see above), and they excite the bipolar and horizontal cells.

(2) **The bipolar cells** : These are 2 types of these cells : *depolarizing and hyperpolarizing* (the latter is probably produced by effect of the horizontal cells). They transmit signals from the rods and cones and horizontal cells to the amacrine and ganglion cells.

(3) **The amacrine cells** : These are mostly *depolarizing cells* (but some conduct signals by producing action potentials). They *transmit signals from the bipolar cells to the ganglion cells, leading to their excitation*. There are *several types of these cells that exert different functions*. One type responds strongly *at the onset of a visual signal* but the response dies out rapidly (thus its function is probably to send strong signals to the brain to indicate sudden changes in light intensity). Another type responds strongly *at the offset of a visual signal*, and still another type responds to a movement of a spot across the retina in a specific direction (*directional sensitive cells*). Therefore, the amacrine cells can be considered as types of interneurons that begin analysis of the visual signals before leaving the retina.

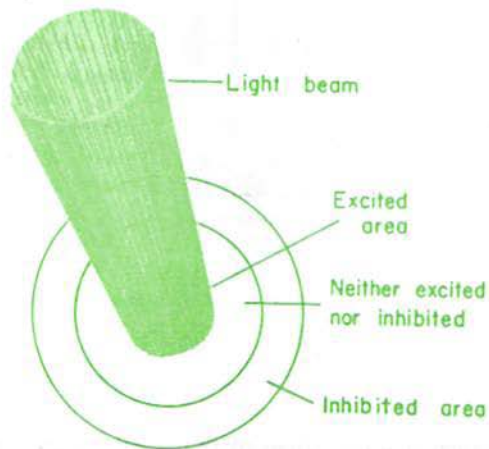


Figure 29 : Excitation and inhibition of a retinal area by a beam of light.

(4) **The horizontal cells** : These are *hyperpolarizing*, and they transmit signals *from the rods and cones to the bipolar cells* in the surrounding area where they cause **lateral inhibition**. Such function is important for :

a) **Detection of the visual contrast** : When a beam of light is focused on the retina, only a small localized area of the retina is excited while the surrounding area is inhibited (figure 29). This prevents spreading of the excitatory signal in the retina, and allows high accuracy in transmitting the contrast borders of the visual images (which is essential for acute vision).

b) **Detection of colours** : Some ganglion cells are excited by only one colour type of cones but inhibited by a second type (through a horizontal cell or a hyperpolarizing bipolar cell) specially in case of red & green cones. Such *reciprocal excitation-inhibition relationship* between colours is essential to produce colour contrast, and it represents a mechanism by which the retina begins to differentiate various colours (therefore *the process of colour analysis begins in the retina and is not entirely a function of the brain*).

(5) **The ganglion cells** : These cells receive signals from the bipolar and amacrine cells and transmit impulses (action potentials) through their axons. The latter constitute the *optic nerve fibres*, and terminate at the lateral geniculate body (or nucleus) in the thalamus. They continuously discharge even when unstimulated in the dark (during which they discharge at rates varying between 5 and 40 per second). There are **3 types of the ganglion cells** :

(a) **W cells (40%)** : These cells are small, transmit *rod vision*, and terminate diffusely in the lateral geniculate body.

(b) **X cells (55%)** : These cells have a medium diameter, transmit *cone vision* (acute and colour vision) and they terminate mainly at the *Parvocellular portion* of the lateral geniculate body (see later), so they are also called the **P cells**.

(c) **Y cells (5%)** : These cells are large and function as the *amacrine cells*, responding to rapid changes in the visual image (either rapid movements or rapid changes in light intensity), and they terminate mainly at the *Magnocellular portion* of the lateral geniculate body (see later), so they are also called the **M cells**.

Synaptic mediators (neurotransmitters) in the retina

A great variety of transmitters are normally released in the retinal synapses, but the *significance of many of them is unknown*. They include : (1) Acetylcholine (2) Dopamine (3) Serotonin (4) GABA and glycine (5) Substance P (6) Somatostatin (7) Enkephalins and B-endorphin (8) CCK and VIP (9) Neurotensin and glucagon (10) TRH & LHRH (see endocrines).

The *amacrine cells are the only retinal cells that liberate acetylcholine*, but there are also dopaminergic and serotonergic amacrine cells, each having a different shape, and probably also a different function (see above).

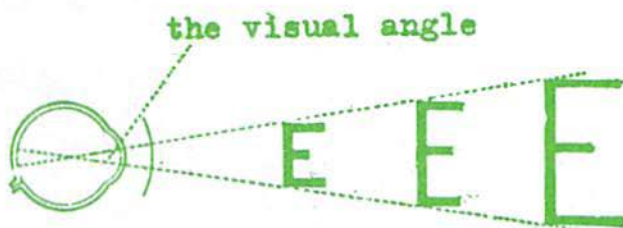


Figure 30 : The size of the letter (E) at various distances from the eye corresponding to a visual angle of one minute.

VISUAL ACUITY

This is the degree of perception of the details and contours of objects by the eye. It is usually determined by finding the *minimum separable* (= the shortest distance by which 2 lines can be separated and still seen as 2 lines).

Normally, the detection of 2 adjacent lines (or objects) requires that their retinal images should stimulate *2 non-adjacent cones*. This is explained as follows : The light rays from 2 adjacent objects form an angle at the nodal point of the lens known as the *visual angle* (figure 30). In the normal eye, the minimal visual angle produced by light rays from 2 adjacent objects

at which the images of the 2 objects are perceived as two is one minute (= $1/60$ degree). In this case, the distance between the retinal images of the 2 objects is about 2 microns, which indicates that 2 foveal cones are stimulated separated by *one unstimulated cone* in between (since the diameter of each foveal cone is about 1.5 microns).

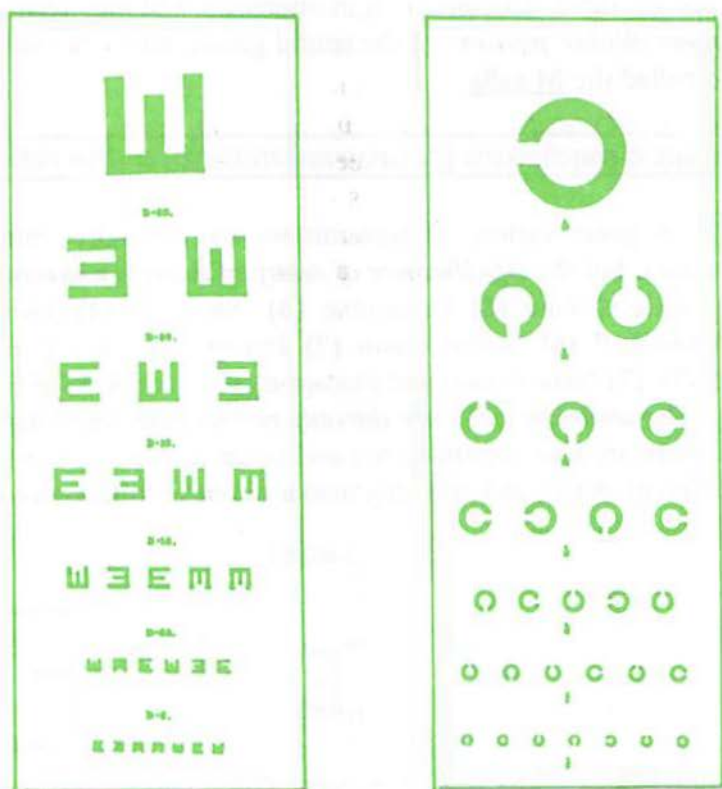


Figure 31 : 2 types of the Landolt's chart for testing visual acuity.

Based on this fact, the various charts used for clinical determination of the visual acuity are constructed. The commonest of these charts are the Snellen's and Landolt's charts, which depend on reading letters drawn in black on a well-illuminated white background (figure 31).

The test chart is placed at the *level of the patient's eyes* and at a distance of 20 feet or 6 meters (so that accommodation is relaxed), and *each eye is tested separately*. It consists of 7 rows of letters, and the spaces between the broken ends of the letters at each row correspond to a visual angle

of one minute at a certain distance from the eye (which is indicated below the row). The uppermost letters are of such a size that they can be read by the normal eye at a distance of **60 meters**, while the following rows (from above downwards) can be normally read at distances of **36, 24, 18, 12, 9 and 6 meters respectively**.

The patient is tested on the chart from above downwards, and the smallest row of letters he can distinguish is determined. The result is expressed as a fraction, the **numerator of which is 6** (the distance between the patient and the chart) while its **denominator is the distance at which the smallest letters distinguished by the person can be seen by the normal eye** e.g. if the person was able to see only the letters that a normal eye can see at a distance of 12 meters, then his visual acuity will be 6/12. The normal visual acuity is 6/6 (or 20/20 if the distances are measured in feet). Therefore, the visual acuity is expressed as a mathematical fraction that determines the **ratio of 2 distances** (which is also the ratio of one's visual acuity to that of a normal person).

Factors that affect the visual acuity

(1) **Age** : The visual acuity is most sharp in children, then it gradually declines till it becomes markedly decreased in old age due to presbyopia.

(2) **The refractive power of the eye** : The visual acuity is decreased if there are errors in the refractive system of the eye e.g. due to myopia, hypermetropia or astigmatism (which are the commonest causes of reduction of the visual acuity).

(3) **The degrees of illumination of the test chart and the contrast between the letters and the background.**

(4) **Diseases of the eyes** : Visual acuity is decreased in various diseases of the eye e.g. keratitis (corneal inflammation) iridocyclitis (inflammation of the iris), cataract, glaucoma, retinal detachment and **retinitis pigmentosa** [the latter is a disease characterized by degeneration of portions of the retina together with deposition of excessive amounts of the melanin pigment in the affected areas, and it leads to blindness that usually starts in the peripheral field of vision then gradually extends to involve the central field].

CHAPTER 6

RETINAL CHANGES ON EXPOSURE TO LIGHT

On exposure to light, the retina evidently shows both electrical and photochemical changes.

Electrical changes in the retina (the electroretinogram)

The electrical activity of the retina is recorded by 2 electrodes connected to a galvanometer or a cathode ray oscilloscope. One of the electrodes is placed on the cornea while the other is placed on the back of the eye (figure 32) or, in humans, it can be placed on the skin of the head.

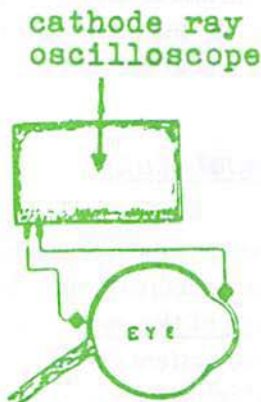


Figure 32 : Recording of potential from the eye.

In the dark, there is a potential difference of about 6 mV between the front and back of the eye (with the front positive). On exposure to light, a series of potential changes occur which are called the *electroretinogram*. It consists of 4 waves called *a, b, c and d waves* (figure 33). The first 3 waves are produced when light is turned on while the last wave is produced when light is turned off. These waves are produced as a result of electrical changes in the various layers of the retina as follows :

(1) ***a wave*** : This is a small negative wave that is probably caused by development of receptor potential in the retinal receptors, and it is not accompanied by discharge of impulses in the optic nerve.

(2) ***b wave*** : This is a large positive wave that is probably caused by activity of the inner nuclear layer of the retina (the bipolar cells, page 29), and it is accompanied by discharge of impulses in the optic nerve.

(3) **c wave** : This is a slowly rising positive wave that sometimes continues after cessation of the light stimulus (figure 33). It is generated in the pigment epithelium of the retina and is not accompanied by discharge of impulses in the optic nerve.

(4) **d wave** : This is a small negative wave that occurs after the light stimulus is cut off. It is probably a *rebound phenomenon in the retina* indicating retinal activity since it is accompanied by discharge of impulses in the optic nerve.

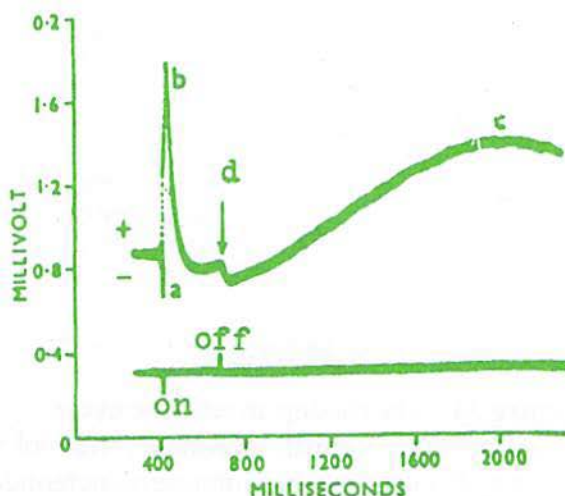


Figure 33 : The electroretinogram.

Photochemical changes in the retina

Light rays cannot directly stimulate the nerve fibres. However, when light is absorbed by the photosensitive compounds (or pigments) present in the photoreceptors, the structure of these compounds changes, and such change triggers a sequence of events that leads to development of the receptor potential. Accordingly, the photoreceptors act as *transducers* that convert light energy into neural activity, and such process of *photo-transduction* involves closure of Na^+ channels in their outer segments (page 39).

The photosensitive pigments are made up of a protein called *opsin* and a carotenoid pigment called *retinene₁* or *retinal* (which is the aldehyde of vitamin A). The pigment in the rods is called *rhodopsin* (or visual purple). Its protein is called *scotopsin*, and in the dark its *retinene₁* molecule is in the *11-cis* form (which has a curved or angulated configuration). The 3 cone colour pigments are almost structurally similar to rhodopsin (and undergo

the same chemical changes described below), except that their proteins are slightly different and are called *photopsins*.

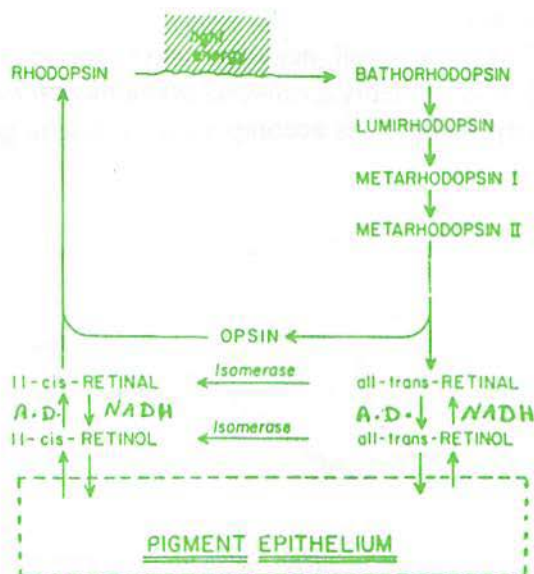


Figure 34 : The rhodopsin retinene cycle.

A.D. = Alcohol Dehydrogenase. Retinal = Retinene₁. Retinol = Vitamin A.
NADH = dihydronicotinamide adenine dinucleotide..

When rhodopsin is exposed to light, it is immediately decomposed. The **only action of light is to change the shape of 11-cis retinene₁**, converting it into the **all-trans isomer** (which has a *straight molecule*), and the resulting compound is called **bathorhodopsin** (which is extremely unstable). It is very rapidly converted to **lumirhodopsin** which is also rapidly converted to **metarhodopsin I** then to **metarhodopsin II**, and finally complete bleaching occurs i.e. separation of all-trans retinene₁ from opsin (figure 34). It is metarhodopsin II (which is also called the **activated rhodopsin**) that initiates the electric changes in rods (page 39).

Reformation of rhodopsin in the dark

Since the 11-cis retinene₁ is the only form that can bind to scotopsin, the all-trans retinene₁ is first re-converted to the 11-cis form (a process that is catalyzed by the **enzyme retinal isomerase**), which then automatically combines with scotopsin to form rhodopsin (figure 34).

Role of vitamin A in rhodopsin formation

A part of the all-trans retinene₁ is reduced by the enzyme alcohol dehydrogenase (in the presence of NADH) to all-trans retinol (which is one form of vitamin A). The latter is then converted to 11-cis retinol (under influence of the retinal isomerase enzyme) which is finally converted to 11-cis retinene₁ (also by effect of the alcohol dehydrogenase and NADH), and this combines with scotopsin forming rhodopsin.

Vitamin A is always present in the rod cytoplasm (and is also *stored in the pigmented layer of the retina*), so it is normally available to supply retinene₁ when required to form more rhodopsin. Accordingly, vitamin A deficiency decreases the formation of rhodopsin, and since the latter is essential for night vision, vitamin A deficiency leads to *night blindness* (= *nyctalopia*). However, this occurs only if vitamin A deficiency lasts for several months, and it can sometimes be completely cured in less than one hour by i.v. injection of vitamin A.

The rhodopsin-retinene cycle (figure 34) *is independent of nervous control*, and all the reactions involved are *independent of light except the conversion of 11-cis retinene₁ into the all-trans isomer* (see above).

Retinal adaptation to varying light intensities**DARK ADAPTATION**

This is the process by which the retina becomes more sensitive in the dark. It occurs when an individual shifts suddenly from bright light to a dim lighted or dark place. The individual at first sees nothing because of the markedly low retinal sensitivity during exposure to bright light, but he gradually begins to identify the outline of objects, though he cannot judge their fine details or colours (= *scotopic vision*), and as time passes his retinal sensitivity is increased and his vision improves.

The visual threshold

This is the *minimal amount of light that produces a light sensation*. It is greatly decreased in the dark (figure 35 left), so the retina becomes able to respond to very faint light. This indicates an enormous increase in the retinal sensitivity, which becomes after 40 minutes of dark adaptation about 25000 times its sensitivity during exposure to light (figure 35 right).

The process of dark adaptation starts soon after entering a dark place. It becomes moderate after 15 minutes and increases to a considerable value within 20-30 minutes, but becomes maximal after one hour or more.

Changes that occur during dark adaptation

(1) **Pupillodilatation** : This allows more light rays to enter the eye, which stimulates a greater number of rods.

(2) **The blue green part of the spectrum becomes the most luminous part** (refer to the Purkinje phenomenon, page 37).

(3) **Regeneration of rhodopsin** : Within 20-30 minutes in the dark, most of the retinene₁ in the rods is converted into rhodopsin, and vitamin A is withdrawn from the pigment cells of the retina to the rods where it is converted into retinene₁, which forms more rhodopsin (page 48). This process continues for several hours (although its rate gradually decreases) as long as the person remains in the dark. *The cone pigments are also regenerated in the dark*, but their regeneration is completed within only 5-10 minutes (see below).

(4) **Marked increase of the retinal sensitivity** : This occurs secondary to regeneration of the photochemical pigments (particularly rhodopsin) and it results in *a marked decrease of the visual threshold*. Such changes are evident in the dark adaptation curve (figure 35) which shows 2 components of the dark adaptation response :

a) During the first 5-10 minutes, there is a rapid (but small) rise of the retinal sensitivity and drop of the visual threshold. This is due to *dark adaptation of the cones* as proved by the fact that when the foveal part of the retina (which contains cones only) is tested for dark adaptation (using a red light), only this part of the curve is obtained.

b) Following the rapid changes in retinal sensitivity and visual threshold, they show similar but slower and much greater changes, which become almost stable after 30 minutes. This is due to *dark adaptation of the rods* as proved by absence of such changes in persons suffering night blindness (in whom rod vision is deficient).

**** Night-fly aviators** (who need maximal visual sensitivity in dim light) often wear red goggles (glasses) when they are in bright light. These goggles allow only red light rays to enter the eyes, which stimulate the rods slightly (so rhodopsin is not bleached) but allow the cones to function normally. Therefore, a person wearing red glasses can see clearly in bright light while his rods are becoming dark-adapted. Accordingly, he avoids waiting at least 20 minutes in the dark to develop maximal dark adaptation.

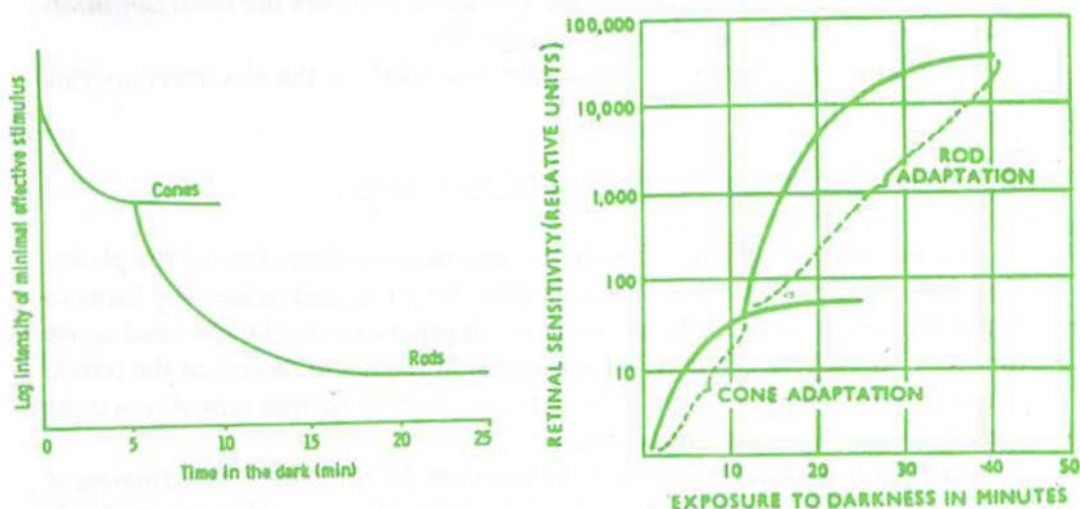


Figure 35 : 2 forms of the dark adaptation curve, showing progressive increased retinal sensitivity (right) and decreased visual threshold (left) on exposure to darkness.

LIGHT ADAPTATION

This occurs when an individual shifts suddenly from a dark place to bright light. At first, the light seems intensely bright and is accompanied by an uncomfortable sensation (so the individual temporarily becomes nearly blind). However, within about 5 minutes, this sensation disappears and acute vision returns and the details of objects and their colours will be clearly seen (= *photopic vision*). Light adaptation is accompanied by a decrease in the retinal sensitivity and an increase in the visual threshold due to reduction of the photochemical pigments in the rods and cones.

Changes that occur during light adaptation

(1) **Pupilloconstriction** : This occurs via the light reflex (page 23), and it reduces the amount of light that enters the eyes, thus decreasing excessive stimulation of the retina (which helps the rapid return of acute vision).

(2) **Bleaching (break down) of the photochemical pigments in the rods and cones to retinene₁ and opsins.**

(3) **Reduction of the retinal sensitivity** : This occurs secondary to reduction of the photochemical pigments in the rods and cones.

(4) *The yellow green part of the spectrum becomes the most luminous part* (refer to Purkinje phenomenon, page 39).

(5) *Electrical changes* : These are recorded as the electroretinogram (page 46).

Vitamins needed for normal retinal functions

(1) **Vitamin A** : This vitamin is essential for formation of the photo-chemical pigments in the rods and cones. Its prolonged deficiency leads to *night blindness* (page 49) as well as degeneration of the rods and cones followed, in severe cases, by degeneration of the neural layers of the retina. Treatment (by supplying vitamin A) can restore normal retinal functions provided its receptors are not damaged.

(2) **Vitamin B** : This vitamin is important for the normal functioning of all neural tissues including the retina. Nicotinamide (a member of vitamin B complex) is particularly important because it is a part of the dihydronicotinamide adenine dinucleotide (NADH) molecule which acts as a coenzyme in the interconversion of retinene₁ and vitamin A in the rhodopsin retinene cycle (figure 34).

CHAPTER 7

COLOUR VISION

Colour vision is the function of the retina *in a light-adapted eye* because it *depends on cones*. The dark-adapted eye sees different colours as grades of grey.

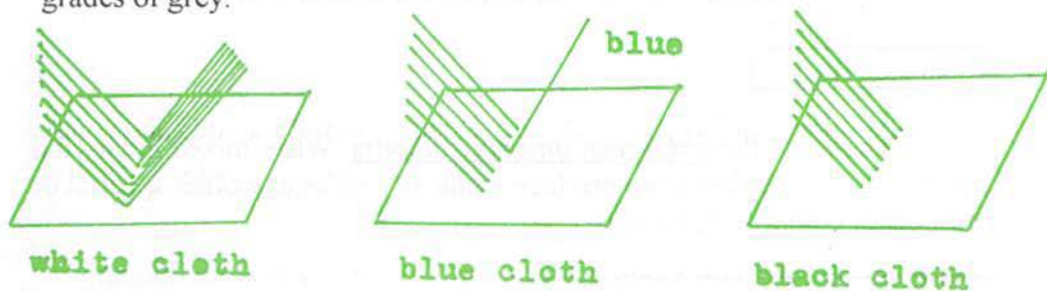


Figure 36 : Reflection and absorption of spectral rays by clothes having different colours.

The visible spectrum in the human eye has a wavelength ranging from about **400 to 725 nm** while the non-visible rays include the following :

a) **Infra-red rays (calorific rays)** : These rays have a longer wavelength than 725 nm and they produce a *thermal (heating) effect*.

b) **Ultra-violet rays (actinic rays)** : These rays have a shorter wavelength than 400 nm and they produce powerful *chemical effects*.

The *normal eye can distinguish the 7 colours of the spectrum as well as about 150 different intermediate colours*. White is the colour at which the 7 spectral colours remain in the same proportion as in sunlight. On the other hand, *black is the sensation produced by absence of light*, and it is probably a +ve sensation because *the blind eye does not see black but sees nothing*.

When a coloured object is illuminated with white light, it reflects some rays and absorbs the others. The colour of a certain object depends on the reflected rays e.g. a blue cloth illuminated with white light looks blue because it reflects the blue rays while the other spectral rays are absorbed. On the other hand, a black object looks so because it absorbs all rays of the spectrum while a white one reflects all of them (figure 36).

The effect of mixing coloured opaque media (e.g. paints or pigments) is quite different from that produced by mixing light rays of different wavelengths e.g. a mixture of blue and yellow paints looks green while a mixture of blue and yellow light rays produces a white light. *Colour mixing in physiology means mixing light rays of different wavelengths*.

Characteristics of colours

- (1) **Hue** : This is the actual colour.
- (2) **Intensity** : This is the amount of light involved.
- (3) **Saturation** : This is the degree of freedom from dilution with white.
A pure colour (not mixed with white) is a *saturated colour*.

Primary colours

These are the red, green and blue colours. When mixed in varying proportions, such colours can produce white as well as any other spectral or extraspectral colour (e.g. purple).

Complementary colours

These are *any pair of colours which when properly mixed produce a sensation of white*. Any colour has a complementary one e.g. yellow is complementary for blue, and red is complementary for green.

After image phenomena

These are visual sensations that are *perceived after removal of visual stimuli*. There are 2 types of after images :

(1) **Positive after image** : After looking at an object for sometime then the *eyes are suddenly closed or turned to a dark surface* (or light is turned off), the image of the object with its original colours is still seen for a brief while. This is due to *after discharge* in the retina as a result of persistence of the chemical changes in the retinal photoreceptors.

(2) **Negative after image** : Looking steadily at a scene having a certain colour and containing bright and dark portions, then *turning suddenly to a bright white surface*, the scene will still be seen for sometime but with the following changes :

a) The *bright portions of the scene appear dark*. This is due to light adaptation of the stimulated retinal areas (so they become less sensitive, and on looking to the white surface they appear dark).

b) The *dark portions of the scene appear bright*. This is due to dark adaptation of the stimulated retinal areas (so they become more sensitive, and on looking to the white surface they appear bright).

c) The *colour sensation appears as the complementary colour of the*

original one in the scene (e.g. if the original colour was yellow, a blue colour will be seen on looking to the white surface). This is due to *adaptation of the retinal receptors to the original colour* (thus on looking to the white surface, its complementary colour only will be perceived).

Flicker and critical fusion frequency (CFF)

Flicker is the sensation of *separate successive visual stimuli* which is produced if a continuous source of light is interrupted (e.g. by a rotating notched disc). Such sensation disappears by increasing the speed of rotation of the disc (so that a continuous sensation of light is perceived). *The frequency of light flashes at which flicker disappears is called the critical fusion frequency (CFF)*. Visual stimuli presented at more than this frequency (e.g. television and motion pictures) *are perceived as a continuous stimulus* (so movies begin to flicker if the projector slows down). This fusion of the successive visual stimuli is due to the property of *after discharge* already described in the positive after image phenomenon (see above).

The CFF is directly proportional to the logarithm of the light intensity (= *Ferry-Porter law*). Thus, if the CFF for a certain light intensity is applied then the light intensity is increased, the sensation of flicker reappears and to abolish such sensation, the CFF should also increase.

Mechanism of colour vision

The Young Helmholtz theory

This is the *most accepted theory of colour vision*. It assumes the presence of *3 kinds of cones*, each of which is *maximally sensitive to only one of the 3 primary colours* (so it is also called the *tricolour mechanism of colour detection*) and that the *sensation of any colour is determined by the relative frequency of impulses discharged from the 3 cone systems*.

Such theory has been proved by chemical identification of 3 different types of cone pigments, which include the following :

- (1) *A blue-sensitive (or short wave) pigment* : This absorbs light maximally in the *blue violet portion of the spectrum* (at a peak wavelength of *440 nm*).
- (2) *A green-sensitive (or middle wave) pigment* : This absorbs light maximally in the *green portion of the spectrum* (at a peak wavelength of *535 nm*).

- (3) A red sensitive (or long wave) pigment : This absorbs light maximally in the *yellow portion of the spectrum* (at a peak wavelength of **565 nm**). This pigment is also sensitive enough in the *red portion of the spectrum* (figure 37).

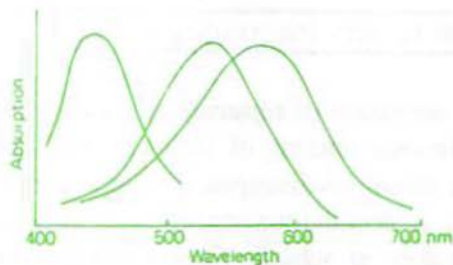


Figure 37 : Absorption spectra of the 3 cone pigments in the human retina.

Interpretation of colour in the C.N.S.

A coloured light stimulates the 3 kinds of cones *unequally* depending on its wavelength. Accordingly, the frequency of impulses discharged from each kind will be different and when these reach the visual cortex, a sensation of the colour will be perceived. This is demonstrated in *the colour sensitivity curves* (figure 38), which show the degree of stimulation of the 3 colour sensitive cones by different lights. For example :

1. An orange light with a wavelength 580 nm stimulates the red cones by an intensity about 99 %, the green cones by 42 % and the blue cones by 0 %. When the ratios **99 : 42 : 0** reach the visual cortex, it interprets the sensation of orange.
2. A blue light with a wavelength 450 nm stimulates the red, green and blue cones by ratios **0 : 0 : 97**, which is interpreted by the visual cortex as a sensation of blue.
3. A ratio of **83 : 83 : 0** is interpreted as yellow.
4. A ratio of **31 : 67 : 36** as green.

In this sense, it can be concluded that colour perception actually starts in the retina (page 42).

****** It is noted that there is *no wavelength of light corresponding to white* (which is a combination of all wavelengths of the spectrum), and approximately *equal stimulation of the red, green and blue cones gives the sensation of white*.

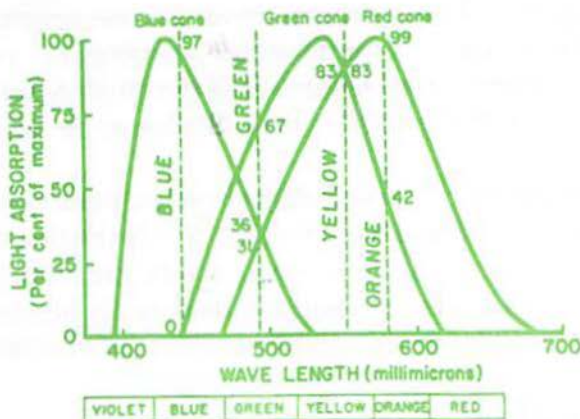


Figure 38 : The colour sensitivity curves.

Colour blindness

Colour blindness may be a complication of certain eye diseases or a manifestation of certain psychological disorders, but commonly it is **inherited as a recessive X-linked characteristic** (due to presence of an abnormal gene on the X chromosome) specially **red blindness** (see below).

Males are more affected than females (8 % and 0.4 % respectively) because in males, colour blindness occurs if their single X chromosome has the abnormal gene while females develop the defect only when both X chromosomes contain the abnormal gene.

As in other diseases that are inherited by males from females (e.g. **haemophilia**), the female children of a colour-blind male are carriers of colour blindness and will pass this defect to their sons.

Types of colour blindness

Weakness of a colour is termed **anomaly** while blindness to a colour is termed **anopia**. On the other hand, the primary colours, (red, green and blue) are referred to by the terms **prot**, **deuter** and **trit** respectively.

According to the Young-Helmholtz theory, colour blind patients are classified into the following 3 types :

(1) **Trichromats** : These patients have the 3 cone systems but one is weak, so they may suffer either **protanomaly** (= weakness of the red colour), **deuteranomaly** (= weakness of the green colour) or **tritanomaly** (= weakness of the blue colour).

(2) **Dichromats** : These patients have 2 cone systems only (i.e. one system is missing), so they may suffer either *protanopia* (= red blindness, in which red is seen green), *deutanopia* (= green blindness, in which green is seen red or blue) or *tritanopia* (= blue blindness, in which blue is seen green).

(3) **Monochromats** : These patients have only one cone system (i.e. 2 systems are missing) or no cone activity at all. In the first condition (*cone monochromatism*), day vision and visual acuity are normal while in the second condition (*rod monochromatism*), there is day blindness in addition to the colour blindness (however, these conditions are extremely rare).

Tests of colour vision

(1) **Colour matching test** : The subject is given a set of coloured wool yarns (or threads) whose colours cover the visual spectrum, and he is asked to select only those which match (i.e. are similar to) a given sample of a particular colour.

(2) **Ishihara's test** : The subject is given special charts called *Ishihara charts* in which there are printed figures (commonly numbers or letters) made up of coloured spots on a background of similar spots that are differently-coloured, and he is asked to identify the figure. The figure's spots are of such colours that a colour blind individual may be confused with the colours in the background spots.

(3) **Edridge-Green lantern test** : This test is specially applied in case of *drivers*. The subject is tested by a lantern situated at a distance of 6 meters. Discs of different colours and others showing different atmospheric conditions (e.g. rain and fog) can be placed at a small illuminated area in front of the lantern's lamp and the subject is asked to differentiate various colours (specially red and green) under different atmospheric conditions.

CHAPTER 8

THE VISUAL PATHWAY

Each half of the retina receives light rays from the opposite side of the visual field i.e. an object placed in the temporal (lateral) side of the visual field projects its rays to the nasal (medial) half of the retina and vice versa (figure 39).

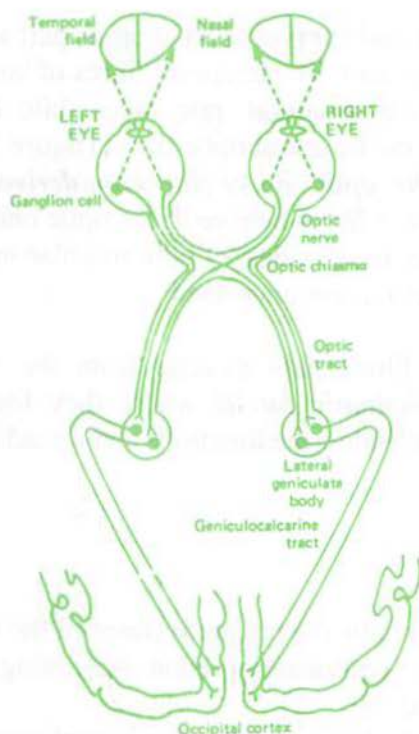


Figure 39 : The visual pathway.

Visual impulses from the photoreceptors first stimulate the bipolar cells of the retina which, in turn, stimulate the ganglion cells, the axons of which form the optic nerve (figure 18). The optic nerve reaches the optic chiasma, where the *nasal fibres of the retina cross to the opposite optic tract, while the temporal fibres proceed to the optic tract of the same side* (figure 39). The fibres then relay in the **lateral geniculate body** from which new fibres arise (= **geniculo-calcarine tract**) and travel in the **optic radiation** (via the posterior limb of the internal capsule) to the **visual cortex at the occipital lobe** where they terminate around the **calcarine fissure** (figure 41).

The optic nerve

Each optic nerve contains about **1.2 million fibres**. It is *not a peripheral nerve but a tract that connects the retina to the lateral geniculate body*. Its fibres contain **no neurolemma** (so they do not regenerate after injury).

The optic chiasma

This receives the optic nerves at its anterior part and the 2 optic tracts project from its posterior part. The temporal fibres of both retinae pass in its lateral parts of each the ipsilateral optic tracts while the nasal fibres cross at its centre to reach the contralateral optic tracts (figure 39).

About **1/3 of the optic nerve fibres are derived from the macular region of the retina**. These fibres behave in the optic chiasma as other fibres i.e. the fibres from the nasal sides of both maculae cross while the fibres from their temporal sides remain uncrossed.

****** Some nerve fibers pass directly from the optic chiasma to the **suprachiasmatic hypothalamic nuclei** where they form connections that synchronize a variety of endocrine functions and circadian rhythms with the light-dark cycle.

The optic tract

Each optic tract contains the temporal fibres of the ipsilateral retina and the nasal fibres of the contralateral retina. According to their *destination*, these fibres are classified into :

- (1) **Visual fibers** (which terminate at the *lateral geniculate body*).
- (2) **Pupillary fibers** (which terminate at the *pretectal area*, page 24).
- (3) **Fibres which terminate at the superior colliculus** from which the *tectospinal and tectobulbar tracts arise* (these tracts mediate the *visuospatial reflexes as well as other reflexes that control posture and equilibrium*).

The lateral geniculate body

This is *one of the thalamic nuclei* and it contains **6 well-defined layers**. The fibres that arise from the nasal side of the contralateral retina terminate at layers 1, 4 and 6, while those arising from the temporal side of the ipsilateral retina terminate at layers 2, 3 and 5 (figure 40).

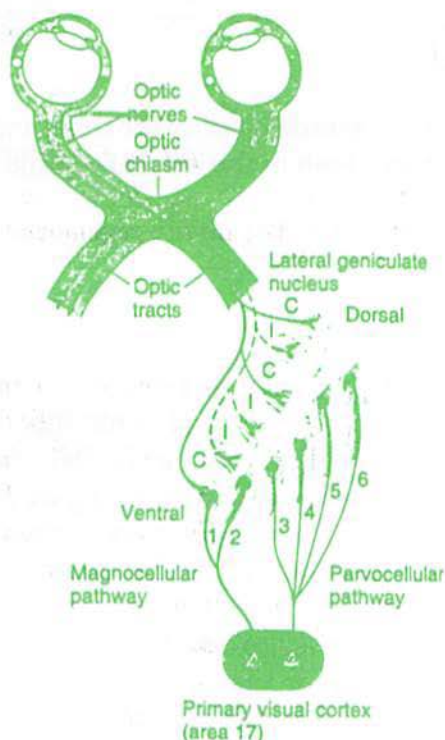


Figure 40 : The lateral geniculate body. C = Contralateral. I = Ipsilateral.

Layers 1 and 2 contain large cells called the **magnocellular cells** while layers 3 – 6 contain small cells called the **parvocellular cells**.

The magnocellular cells receive signals from the large retinal ganglion cells called Y or M cells (page 43), and discharge to the *superficial part of layer 4 C in the primary visual cortex (area 17)*. Such **magnocellular pathway** carries signals for detection of **movement, depth & flicker**.

On the other hand, the parvocellular cells receive signals from the small retinal ganglion cells called X or P cells (page 43) and discharge to the *deep part of layer 4 C in the visual cortex*. Such **parvocellular pathway** carries signals for **colour vision, shape and fine detail**.

Cells in the interlaminar region of the lateral geniculate body receive an input from the P ganglion cells, then project to layers 2 and 3 in the visual cortex (via a separate component of the parvocellular pathway). These layers contain clusters of special cells called **blobs** that are arranged in a mosaic form, and are concerned mainly with **colour vision**.

****** Only 10-20 % of the input to the lateral geniculate body comes from the retina. Major inputs also come from the visual cortex & other brain regions, and are involved in a feedback regulation of the visual input to the cortex.

The optic radiation

This is made up of the *geniculo-calcarine tract*, and it is located in the posterior part of the posterior limb of the internal capsule (refer to C.N.S.)

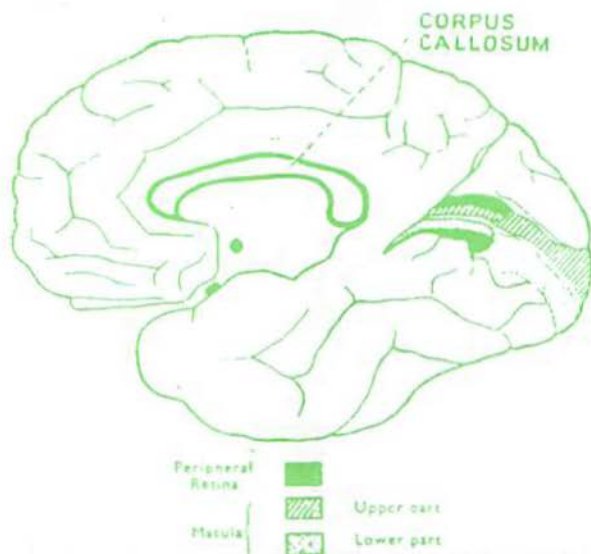


Figure 41 : Medial view of the human cerebral hemisphere showing the visual cortex.

The visual cortex

This is present in *the occipital lobe*, and it receives fibres from the *temporal side of the ipsilateral retina and the nasal side of the contralateral retina*. It contains areas 17, 18 and 19.

(A) **Area 17** : This is the *primary visual centre* and it is also known as the *visuosensory area*. It occupies the *calcarine fissure and the 2 gyri above and below it on the medial surface of the occipital lobe*. In this area, the upper halves of the retinae are represented above the calcarine fissure while the lower halves are represented below it. On the other hand, the peripheral parts of the retinae are represented most anteriorly while the *macular regions are much more widely represented* (occupying the posterior part and the occipital pole). Area 17 performs the following functions :

1. Perception of visual sensations (but without understanding their meaning)
2. Fusion of the 2 images formed on both retinae.
3. Localization of objects in space in relation to each other.
4. Perception of colour vision.

(B) Area 18 : This area is also known as the *visuopsychic area*, and it lies anterior to area 17. It performs the following functions :

1. It recognizes the nature of objects and correlates their colours.
2. It is concerned with interpretation of the visual sensations (so it is also called the *visual association or interpretative area*). Thus, its injury on the left side (in right-handed individuals) leads to *visual aphasia*, in which the patient cannot understand the meaning of written words (refer to C.N.S.).
3. It is concerned with localization of objects in space in relation to the observer (i.e. depth perception).

(C) Area 19 : This area is also known as the *occipital eyefield area* and it lies anterior to area 18. It performs the following functions :

1. It shares area 18 its functions.
2. It sends information to other parts of the cerebral cortex e.g. the frontal eyefield area (area 8) during accommodation (page 15).
3. It exerts *motor functions*, including (a) Conjugate deviation of both eyes to the opposite side (b) Fixation movements by which the eyes are fixed on an object (c) Physiologic nystagmus (see next).

Physiologic nystagmus

This is jerky movements of the eyeballs that normally occur on looking steadily at a stationary object. Such movements are important because *the visual receptors and their neural connections adapt on continuous stimulation* (specially the latter), and this would lead to disappearance of the object's image. However, these movements cause the retinal images to be rapidly and continuously shifted from one receptor to the other, thus the object will be continuously seen.

****** Recent studies have revealed that visual stimuli not only activate the occipital cortex but also parts of the inferior temporal cortex, the postero-inferior parietal cortex and portions of the frontal lobe. Also, the activated subcortical structures are not only the lateral geniculate bodies, but also the superior colliculi, caudate nuclei, pulvinar, putamen, claustrum and the amygdaloid nuclei.



Figure 42 : Effects of various lesions in the visual pathway.

LESIONS OF THE VISUAL PATHWAY

(1) Unilateral lesions in the optic nerve

These lead to *blindness in the corresponding eye* (No. 1 in figure 42). No light reflex is obtained if the blind eye is stimulated, but *miosis occurs in the blind eye if this reflex is initiated from the other eye* (because the oculomotor nerve is intact).

(2) Lesions in the centre of the optic chiasma

These lesions are usually due to *anterior pituitary tumours*. They lead to *bitemporal hemianopia* (i.e. loss of vision in the temporal parts of the visual fields of both eyes) because the crossing nasal fibres of both retinae are injured (No. 2 in figure 42). The light reflex is obtained only from the temporal halves of both retinae.

(3) Lesions in the periphery of the optic chiasma

These lesions are rare, but they can occur as a result of an *aneurysm (severe dilatation) of the internal carotid arteries*. If the lesion is bilateral, it leads to *binasal hemianopia* (i.e. loss of vision in the nasal parts of the visual fields of both eyes) because the temporal fibres of both retinae are injured, and the light reflex is obtained only from the nasal sides of both retinae. On the other hand, if the lesion affects one side only, unilateral nasal hemianopia results.

(4) Lesions in the optic tracts

These lesions cause blindness in the opposite parts of the visual fields of both eyes (No. 3 in figure 42) i.e. ***contralateral homonymous hemianopia*** e.g. a lesion in the right optic tract leads to blindness in the left part of both fields of vision (i.e. the temporal field of the left eye and the nasal field of the right eye). The ***light reflex is lost at the blind sides of both retinae*** and can be obtained only from the nasal half of the ipsilateral retina and the temporal half of the contralateral retina.

(5) Lesions in the lateral geniculate body, optic radiation or the occipital cortex

These lesions also lead to ***contralateral homonymous hemianopia*** (No.5 in figure 42). However, ***the light reflex is still obtained from the blind sides of both retinae*** (because the optic tract is not affected in such lesions).

**** Therefore, the light reflex can differentiate between lesions in the optic tract and lesions beyond it (e.g. in the optic radiation) although both lesions lead to contralateral homonymous hemianopia. It is lost at the blind sides of the retinae in lesions of the optic tract, but remains intact at these blind sides in lesions of the optic radiation.**

**** Less extensive lesions at the occipital lobe or the optic radiation often lead to incomplete contralateral blindness e.g. *contralateral homonymous quadrantanopia* (No. 4 in figure 42)**

**** The sites of various lesions in the visual pathway can be diagnosed by determining the defects that occur in the visual fields (which are characteristic in each lesion). Visual field determination is commonly performed by *perimetry* (see next chapter).**

Sparing of the fovea (= macular sparing)

Unilateral lesions of the occipital lobe lead to contralateral homonymous hemianopia (see above), but macular vision is usually selectively spared i.e. not affected (No. 6 in figure 42) because :

(1) The macular region in the visual cortex receives blood supply from 2 sources (*the middle and posterior cerebral arteries*) while other areas are supplied by the posterior cerebral artery only. So thrombosis of the posterior cerebral artery (which is the commonest cause of occipital lobe lesions) usually affects most of the visual cortex but spares the macular region.

(2) The macula is *widely represented in the visual cortex* and the area of its representation is separate from that of the peripheral parts of the retina (so occipital lobe lesions often spare it or may not destroy it completely).

CHAPTER 9

VISUAL FIELDS, BINOCULAR VISION & EYE MOVEMENTS

The visual field of an eye is the maximal area of space that can be seen by this eye. It is **not circular** because it is cut off medially by the nose, superiorly by the roof of the orbit (and eyebrows) and downwards by the cheeks (so its nasal side is smaller with an irregular outline while its temporal side is larger with a regular outline). Normally, the visual field of each eye extends about *50 degrees upwards*, *80 degrees downwards*, *60 degrees nasally* and *100 degrees temporally* (figure 43).

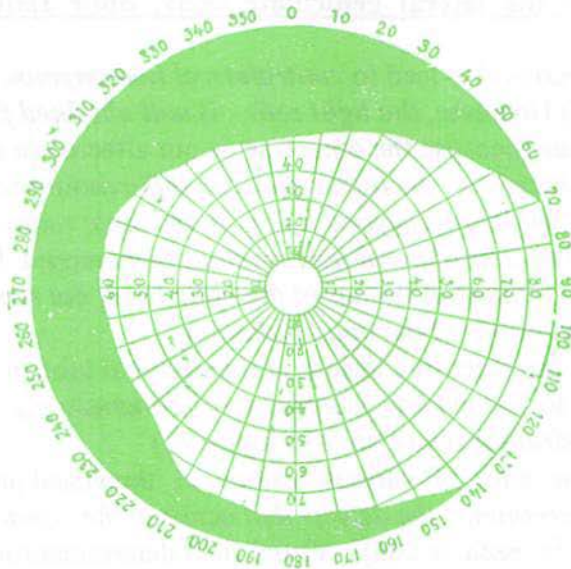


Figure 43 : A perimetry chart showing the visual field of the right eye.

Measurement of the visual fields

The visual fields can be mapped by an instrument called the *perimeter* (figure 44) and the process of measurement is called perimetry (figure 45). The perimeter consists of a metal arc shaped like half of a circle, the centre of which is marked by a white spot or a mirror. **One eye is examined at a time while the other is covered.** The subject's head is supported on a chin-rest and he is asked to fix the examined eye on the centre of the perimeter. The examiner then moves a white target along the arc (starting from its periphery towards its centre) and the subject indicates when the target is first seen (figure 45). This process is repeated in various meridians (by rotating the arc around its centre). The points at which the target is first seen

in different meridians are recorded upon a special chart and the line joining these points forms the boundary of the visual field (figure 43).

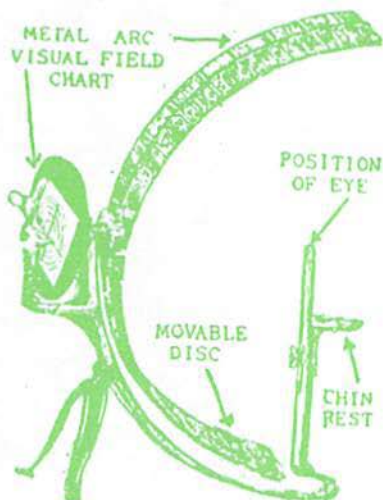


Figure 44 : The perimeter

****** In the retina, *the visual field is reversed and inverted*. Consequently, a lesion in right area 17 above the calcarine fissure (which receives fibres from the upper right quadrants of both retinae) leads to left lower homonymous quadrantanopia, while a lesion in the same area but below the calcarine fissure leads to left upper homonymous quadrantanopia.

****** The visual field can also be determined by the *confrontation test*. In this test the patient's field is compared with that of the examiner (who is supposed to be normal). The patient sits in front of the examiner at a distance of about **60 cm**. To examine the patient's right eye, he is asked to close his left eye and to keep his right eye fixed on the examiner's left eye. The examiner closes his right eye then he approaches his outstretched arm (while moving a finger) from the periphery inwards, and the patient indicates when he first sees the examiner's finger. This process is performed in various meridians.

Importance of determination of the visual fields

- (1) It greatly helps in *localization of the sites of lesions in the visual pathway* (page 65).
- (2) It localizes the *sites of scotomata* (blind spots) in the retina. The optic disc is a physiologic (normal) blind spot (page 32).
- (3) It helps in diagnosis of some retinal diseases e.g. retinitis pigmentosa.



Figure 45 : Measurement of the visual field by perimetry.

BINOCULAR VISION

This is seeing an object *with the 2 eyes at the same time*. It is due to overlap of the central parts of both visual fields (figure 46), so objects placed in the overlapping area form images on the retinae of both eyes. Such overlap (and accordingly, the area of binocular vision) is most apparent in man and monkeys (figure 47 a).

In some animals (e.g. rabbits) the overlapping area is very small (figure 47 b) because their eyes are almost laterally-placed in the head. Consequently, the vision in these animals is *mostly monocular*, and their areas of binocular vision are much limited.

The *monocular field of vision in man consists of a crescentic area (about 35 degrees) at the outer part of each temporal field* (figure 47 a).

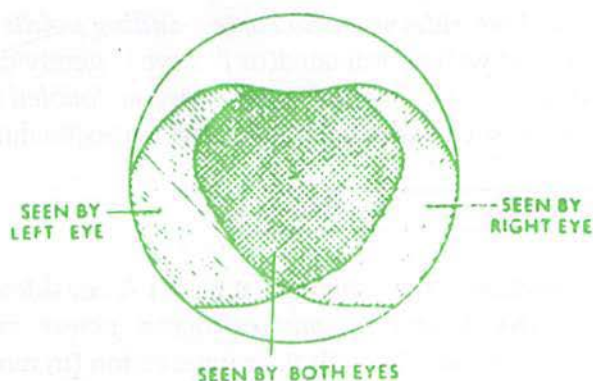


Figure 46 : Overlap of the central parts of both visual fields.

Light rays from objects placed in these regions fall only on the nasal side of the retina of the nearer eye but not on the temporal side of the contralateral retina *because the rays are shaded by the nose*.

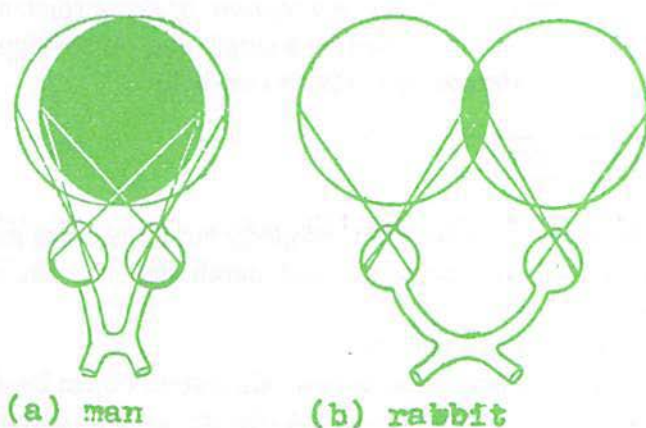


Figure 47 : Areas of binocular vision (black) and monocular vision (white) in man and rabbits.

FUSION

The 2 images of an object placed in the area of binocular vision (one from each retina) are fused at the cortical level (area 17) into a single image. The retinal points at which the images of an object must fall to be seen binocularly as a single image are called the corresponding points. They are located at the nasal side of one retina and the temporal side of the other. The

foveae centralis at both sides are also corresponding points. If the external ocular muscles are not well coordinated (or the eye is gently displaced by the finger), **diplopia** occurs i.e. *the object's image is doubled* (double vision) because the images in such cases fall outside the corresponding points.

Factors that affect binocular vision

For proper binocular vision, there must be (a) A considerable *overlap* of both visual fields (b) A nearly *equal refractive power* in both eyes (c) *Normal external ocular muscles* with their innervation (to move the eyeballs so that both images of an object *fall at the corresponding points* of both retinae) (d) *An intact visual cortex* (as fusion of images occurs in area 17).

Advantages of binocular vision

(1) It gives *compensation for a weak eye* e.g. if there are defective spots in the retina of one eye, they can be masked through binocular vision.

(2) It provides *more accurate perception of depth* (distance of objects from the eyes) than that provided by a single eye. Accordingly, binocular vision is important for *stereoscopic vision* (see below).

Perception of depth

This is principally a monocular property but it becomes *more accurate by binocular vision*. The perception of depth depends on the following factors (figure 48) :

- (1) The relative sizes of objects.
- (2) The blocking or occlusion of part of a distant object by a nearer one.
- (3) The fade of colour and details of objects as their distances from the eye are increased.
- (4) The distribution of light and shade on the surfaces of objects.
- (5) The shadows which an object casts upon its surroundings.
- (6) The *perspective* (i.e. parallel lines appear to converge with distance).
- (7) *Movement parallax* : In case of moving objects, their movements relative to each other help in judging their distances from the eye. Also, when the head moves in a certain direction, the nearer objects move in the opposite direction while distant objects move in the same direction. These apparent movements are known as the movement parallax, and they greatly contribute in the perception of depth.

When we look at some object or scene the view seen by the RIGHT EYE is slightly different from the view seen by the LEFT EYE.

These two DISSIMILAR RETINAL IMAGES are fused in the Visual Centres of the Brain to give a 3-dimensional picture – an appreciation of DEPTH as well as of HEIGHT and WIDTH.

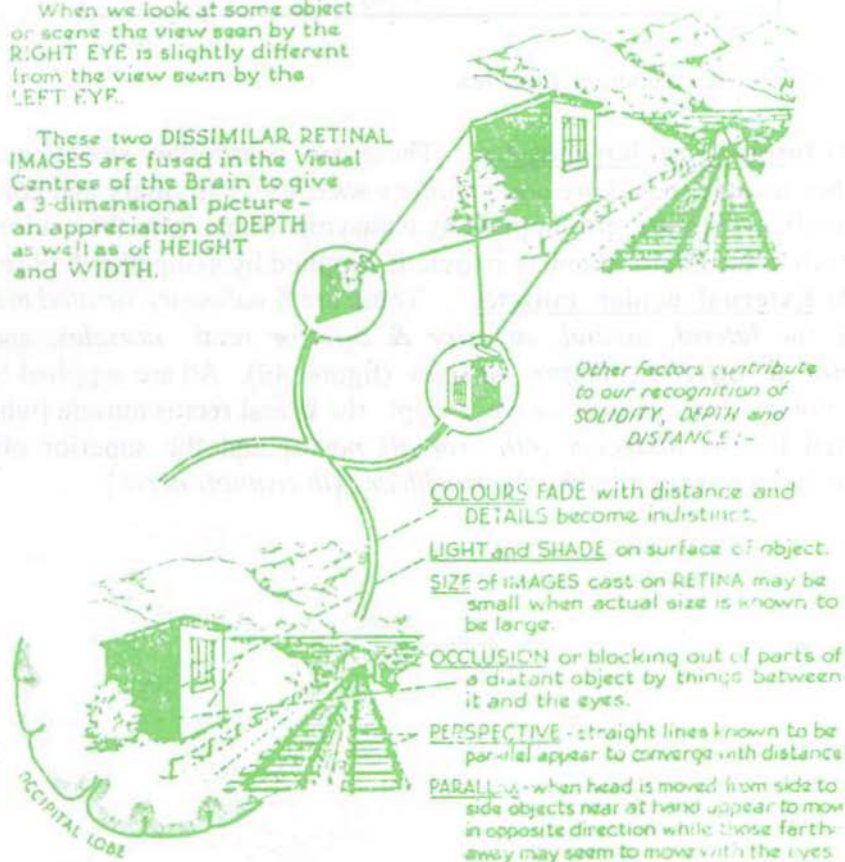


Figure 48 : Various factors involved in the perception of depth and stereoscopic vision.

Stereoscopic vision

This depends on determination of the 3 dimensions of objects (height, width and depth). It can be perceived by monocular vision but it is better recognized by binocular vision (due to the more accurate perception of depth). In addition, the corresponding points of both retinae form *slightly dissimilar images of the object* (figure 48) because the 2 eyes are few centimeters apart from each other. Such dissimilar images fuse in the visual cortex producing a feeling of stereoscopic vision. Accordingly, the main factors that affect stereoscopic vision are (a) All factors that determine the perception of depth (see above) (b) The formation of dissimilar images on both retinae.

Ocular muscles and eye movements

There are 2 types of ocular muscles :

(1) **Internal ocular muscles** : These are *involuntary smooth muscles* and they include the *ciliary and pupillary muscles*. The ciliary and constrictor pupillae muscles are supplied by parasymp. fibers from the oculomotor nerve while the dilator pupillae muscle is supplied by sympathetic fibres.

(2) **External ocular muscles** : These are 6 *voluntary striated muscles* called the *lateral, medial, superior & inferior recti muscles*, and the *superior & inferior oblique muscles* (figure 49). All are supplied by the *oculomotor (3rd cranial) nerve* except the lateral rectus muscle [which is supplied by the *abducent (6th cranial) nerve*] and the superior oblique muscle [which is supplied by the *trochlear (4th cranial) nerve*].

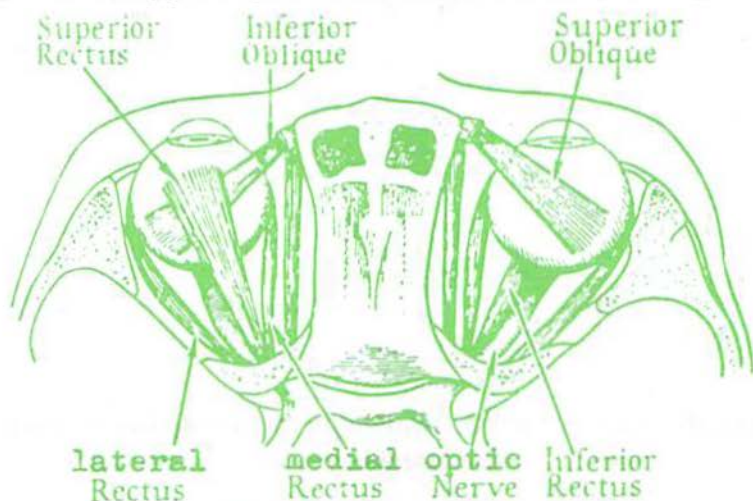


Figure 49 : The external ocular muscles.

Movements of the eyeballs

The external ocular muscles rotate the eyeball around **3 axes** : (a) A **vertical axis** (for adduction and abduction movements) (b) A **horizontal (antero-posterior) axis** (for torsional movements) (c) A **transverse axis** (for upward and downward movements). They act in harmony, so that their contraction produces *conjugate deviation of both eyes* (i.e. movement of both eyes in the same direction). This leads to falling of the images at the

corresponding points and production of a single visual sensation. Their contraction follows the rule of *reciprocal innervation* (i.e. when a muscle contracts, its antagonist relaxes and vice versa).

Nervous control of eye movements

Conjugate movements of the eyes are controlled by the cortical areas **8 and 19** (frontal and occipital eye-field areas respectively). These areas regulate the eye movements through their control on the *medial longitudinal bundles* (which connect the nuclei of the 3 cranial nerves that innervate the external ocular muscles). These bundles are also controlled by signals from the vestibular nuclei, cerebellum, spinal cord and the superior colliculi.

Stimulation of *area 8 produces conjugate deviation of both eyes to the opposite side*. On the other hand, it is believed that *area 19 receives information from areas 17 and 18 about the fusion of images formed on both retinae*. If they are not properly fused (i.e. the images are not in the exact corresponding points), it sends *directly or through area 8 to the medial longitudinal bundles* to adjust the activity of the external ocular muscles, so as to bring the images at the corresponding points.

The superior colliculi

These are the *chief centres for the visual reflexes* which include :

- (1) Movement of the eyes to keep them fixed on an object when the head is turned in the opposite direction.
- (2) Movement of the head and eyes to keep a moving object in view.
- (3) Movement of the limbs, neck and trunk muscles as a defensive response against harmful stimuli (e.g. a blow threatening the eyes).

This is called the *visuospinal reflex* (refer to C.N.S.).

These reflexes are mediated through afferent impulses that reach the superior colliculi from the visual cortex, or the retina via the optic tract (page 60), and efferent impulses that arise from them which regulate :

(a) The eye movements (through controlling the medial longitudinal bundles)

(b) The contraction of the muscles of the limbs, neck and trunk (through the tectospinal and tectobulbar tracts, refer to C.N.S.).

Strabismus (= squint)

This is an eye disorder caused by *incoordination of the external ocular muscles* (commonly due to paralysis of some of these muscles secondary to injury of their nerve supply). Also *untreated hypermetropia in children may lead to convergent squint* (due to persistent use of accommodation).

The conjugate movements are lost, so the visual axes become no longer maintained in a position that brings the images at corresponding points resulting in *diplopia*. In children under the age of 6 years, when the visual images chronically fall outside the corresponding points, one of the images is eventually suppressed by the visual cortex (= *suppression scotoma*) and diplopia consequently disappears (but this does not occur in adults). However, if the condition is not properly or early treated (before the age of 6 years), permanent loss of visual acuity occurs in the eye that generates the suppressed image (a condition called *amblyopia ex anopsia*).

Treatment of strabismus

Squint can be corrected (or improved) by one of the following procedures :

- (1) Surgical shortening of some of the external ocular muscles.
- (2) Certain eye-muscle training exercises.
- (3) Using glasses with special prisms that bend the light rays sufficiently to compensate for the abnormal position of the eyeball.
- (4) In hypermetropic children, the early use of convex lenses may prevent development of convergent squint.

CHAPTER 10

THE AQUEOUS HUMOUR and INTRAOCULAR PRESSURE

THE AQUEOUS HUMOUR

The aqueous humour is an *alkaline clear fluid which fills both the anterior and posterior chambers of the eye*. It has a high O_2 tension and contains $NaCl$ and HCO_3^- (the formation of the latter is helped by the *carbonic anhydrase enzyme*) as well as a sugar, a small amount of protein and hyaluronic acid.

Mechanism of formation of the aqueous humour

The aqueous humour is formed at a rate of $2-3 \text{ mm}^3 / \text{minute}$ mainly by the epithelium of the *ciliary processes of the ciliary body*. Na^+ is first *actively secreted* by the ciliary epithelium, and this is followed by passive diffusion of Cl^- , HCO_3^- and water by osmosis.

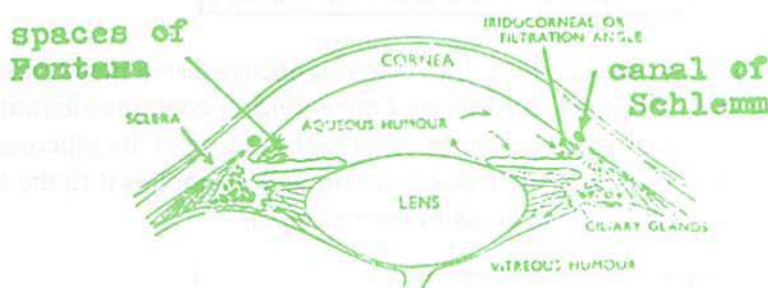


Figure 50 : Circulation and drainage of the aqueous humour

Circulation and drainage of the aqueous humour

The aqueous humour enters the posterior chamber and flows through the suspensory ligament of the lens, then it reaches the anterior chamber through the pupil. The fluid then flows towards the *iridocorneal angle* (= *anterior chamber angle or filtration angle*) where it passes through the *spaces of Fontana* and drains into the *canal of Schlemm* (figure 50). This canal is a thin-walled venous channel that is extremely permeable (even to large protein molecules). From the canal of Schlemm, the aqueous humour drains into the *aqueous veins then finally into the extraocular veins*.

Movement of the aqueous humour

The aqueous humour moves by the following mechanisms :

(1) ***Intermittent variation in the intraocular pressure*** : This is produced by variations in the arterial blood pressure during the cardiac and respiratory cycles (see below).

(2) ***The continuous movements of the eye lids.***

(3) ***Convection currents in the aqueous humour*** : These occur because the temperature of the iris is $3-5^{\circ}\text{C}$ higher than that of the cornea.

Functions of the aqueous humour

(1) It gives ***nutrients to the avascular structures*** (lens and cornea).

(2) It ***maintains the intraocular pressure*** (see below).

(3) It plays a role as ***a refractive medium in the eye*** (page 3).

(4) It is a route for ***removal of waste products from the eye.***

THE VITREOUS HUMOUR

This is a transparent jelly-like material enclosed in a membrane that ***occupies the space between the lens and the retina.*** It contains albumin and hyaluronic acid (which is responsible for its high viscosity). Its glucose content is less than that of the aqueous humour, and it supplies it to the retina (which adds lactic and pyruvic acids to the vitreous).

Functions of the vitreous humour

(1) It maintains the ***globular shape of the eyeball.***

(2) It ***transmits pressure*** from the choroidal vessels to the anterior compartments of the eye (see below).

(3) It ***supports the retina and supplies it with glucose*** (see above).

(4) It has a ***little role as a refractive medium in the eye*** (page 3).

THE INTRAOCULAR PRESSURE (IOP)

The IOP is normally ***12-20 mmHg (average 15 mmHg)*** and it shows a ***diurnal variation up to 4 mmHg*** (being highest in the morning and lowest in the evening). It falls to about 8 mmHg after death due to arrest of the ocular circulation. It is ***maintained nearly equal to the choroidal capillary***



Figure 51 : Measurement of the intraocular pressure by the tonometer.

blood pressure (so if the latter increases, e.g. by coughing or straining, the IOP also increases and vice versa). It is *kept fairly constant* in spite of the pulsatile and respiratory variations in the arterial blood pressure. Clinically, it is measured by an apparatus called the *tonometer* (figure 51), which is applied to the cornea (after anaesthetizing it by cocaine drops). It can also be roughly assessed by palpating the eyeball with the fingers (figure 52).



Figure 52 : Assessment of the intraocular pressure by palpating the eyeball.

Regulation of the IOP

The IOP is regulated *mainly by controlling the outflow of the aqueous humour*. If the IOP increases, the spaces of Fontana are distended, allowing an increase in the outflow of the aqueous, so the IOP decreases to its normal level. On the other hand, a decrease in the IOP produces an opposite effect i.e. the spaces of Fontana are closed, so the outflow of the aqueous humour is decreased and consequently the IOP increases to its normal level.

Importance of the IOP

The IOP is important for the *normal focusing mechanism of the eye*. If the IOP decreases, the suspensory ligament of the lens relaxes and the lens becomes more spherical, resulting in an increase in its power (so images are focused in front of the retina as occurs in myopia). On the other hand, if the IOP increases, the suspensory ligament is stretched and, consequently, the accommodation mechanism for near vision will be impaired.

GLAUCOMA

This is a serious disease in which the IOP markedly increases (up to 60-70 mmHg). It is due to either (a) Obstruction of the canal of Schlemm (b) Decreased permeability through the trabeculae of the spaces of Fontana due to an inflammatory process (open-angle glaucoma) (c) Infections of the iris that cause its sticking to the lens (d) Forward movement of the iris, which may obliterate the iridocorneal angle (angle-closure glaucoma). The increased IOP leads to *headache and severe eye-pain, mydriasis, misty vision, impairment of the focusing mechanism of the eye, upping of the optic disc and pressure on both the retinal vessels and optic nerve fibres against its rim (which may finally lead to blindness)*.

Treatment of glaucoma

(1) Inducing *pupilloconstriction by parasympathomimetic drugs* (e.g. eserine or pilocarpine drops). This opens the spaces of Fontana and increases the outflow of the aqueous humour (which decreases the IOP).

(2) Giving *drugs that decrease secretion of the aqueous humour* e.g. *diamox* (which acts through inhibiting the carbonic anhydrase enzyme).

(3) Surgery is indicated if the above medical treatments fail. Usually a *hole (trephine) is made at the iridocorneal angle* (to increase the outflow of the aqueous humour).

SECTION II

PHYSIOLOGY OF HEARING

CHAPTER 1

STRUCTURE OF THE EAR & PHYSICS OF SOUND

Functions of hearing

- (1) It is the principal means of establishing relations and transmitting thoughts among human beings.
- (2) It is essential for speech and learning (refer to C.N.S.).

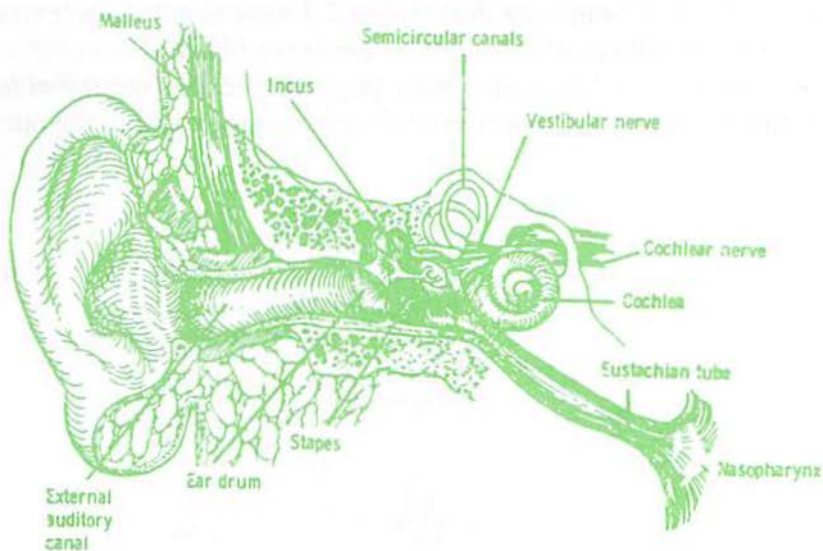


Figure 53 : Structure of the human ear

Structure of the human ear

The human ear consists of **3 parts** (figure 53) :

- (1) **External ear** : This consists of the following structures :

- a. The *ear pinna* (or auricle).
- b. The *external auditory meatus* (a narrow canal that conducts the sound waves inwards to the tympanic membrane).
- c. The *tympanic membrane (or ear drum)* which separates the external ear from the middle ear.

(2) **Middle ear** : This is an air-filled cavity in the temporal bone that is *connected to the nasopharynx by the Eustachian tube*. It contains the following structures :

- a. *3 small bony ossicles* (called from out inwards, the *malleus, incus and stapes*).

b. 2 nerves (the facial nerve and the chorda tympani).

c. 2 skeletal muscles called the tensor tympani and stapedius muscles.

The former is supplied by a branch from the trigeminal (5th cranial) nerve while the latter is supplied by a branch from the facial (7th cranial) nerve.

d. 2 foramina, which separate the middle ear from the internal ear.

These are the *foramen ovale* (or oval window) and the *foramen rotundum* (or round window).

(3) **Internal ear (= cochlea or auditory labyrinth)** : This is a coiled bony tube about 35 mm long that makes $2\frac{3}{4}$ turns around a central bony core called the *modiolus* which contains the nerve fibres of the cochlear (8th cranial) nerve (figure 54). A thin bony projection (called the *spiral lamina*) winds round the modiolus, and from it 2 membranes extend to the outer wall

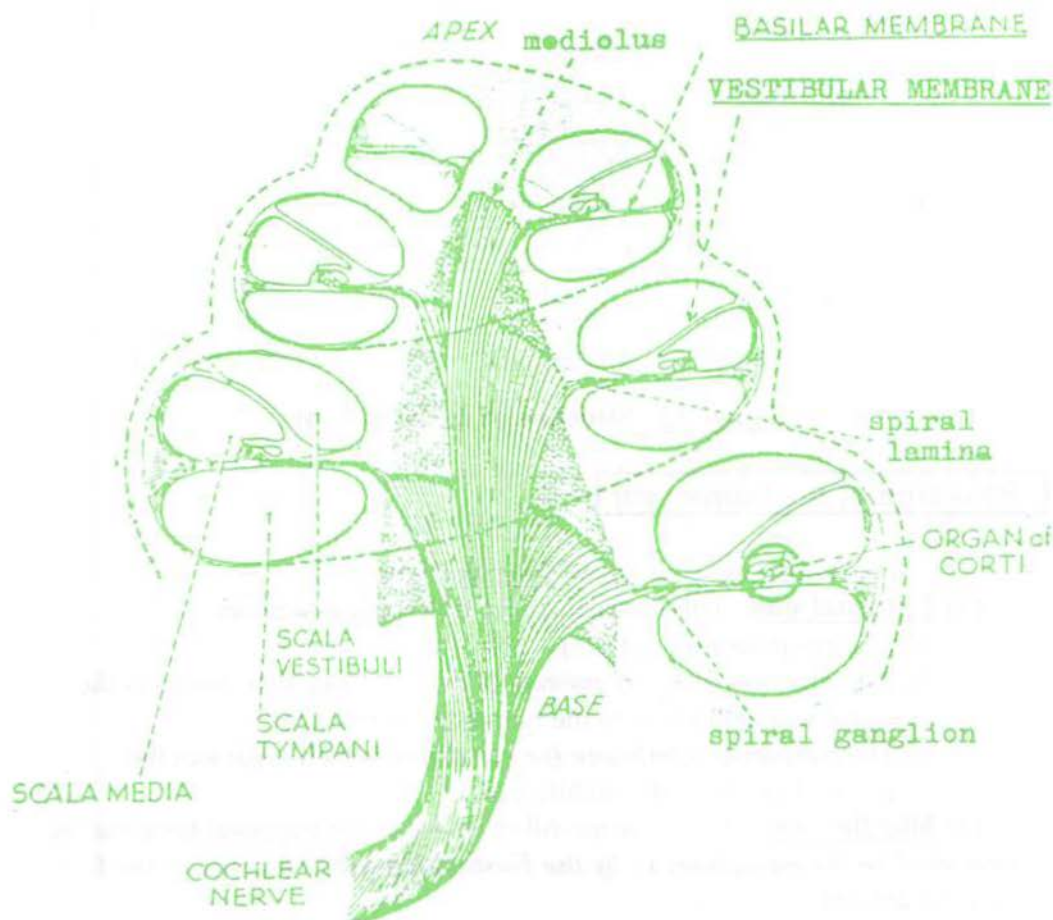


Figure 54 : Structure of the cochlea (a longitudinal section).

of the cochlea. These are called the *vestibular (or Reissner's)* and the *basilar membranes*, and they divide the cochlea into 3 compartments (or *scalae*) : *scala vestibuli*, *scala media* and *scala tympani* (figure 54). The *scala media* is filled with *endolymph*, and it ends at the apex of the cochlea as a blind sac, but it communicates at its base with the vestibular apparatus (= the nonauditory labyrinth) which is formed of the utricle, saccule and the semicircular canals (refer to C.N.S).

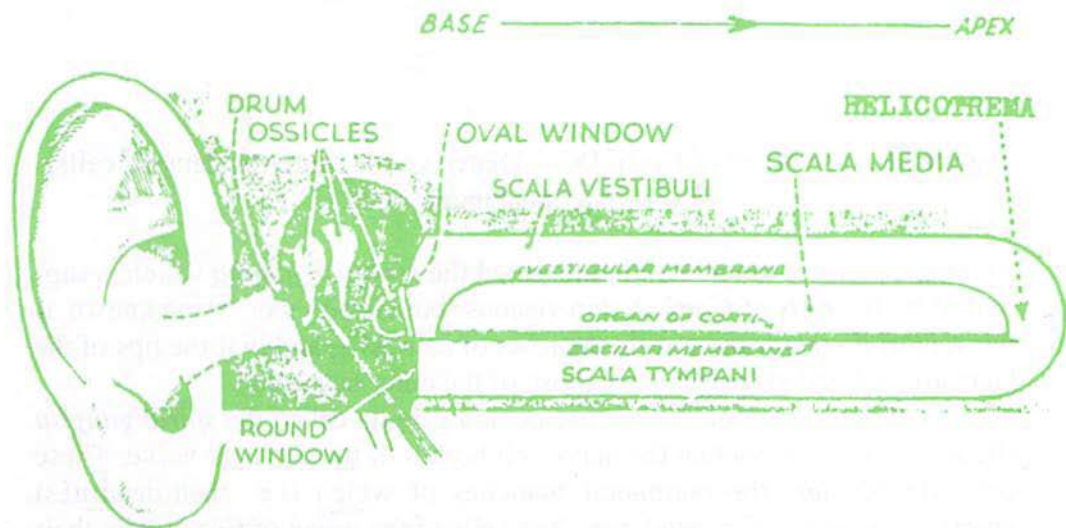


Figure 55 : Structure of the cochlea (unfolded).

Both *scalae vestibuli* and *tympani* contain *perilymph* and communicate with each other at the apex of the cochlea through a small opening called the *helicotrema*. At the base of the cochlea, the *scala vestibuli* ends at the *foramen ovale* (which is closed by the foot plate of the stapes) while the *scala tympani* ends at the *foramen rotundum* which is closed by a flexible *secondary tympanic membrane* (figure 55).

The organ of Corti

This is the sense organ of hearing. It lies on the *basilar membrane*, and it extends in a *spiral shape* from the *base* to the *apex* of the cochlea. It is formed of 2 sets of *hair cells (the auditory receptors)* that rest on supporting (or phalangeal) cells and are separated by the *tunnel of Corti* (figure 56). In each cochlea, there are about 3500 inner hair cells (arranged in one row) and 20000 outer hair cells (arranged in 3 rows). There are tight junctions between the hair cells and the adjacent phalangeal cells (which prevent the endolymph from reaching the bases of the hair cells).

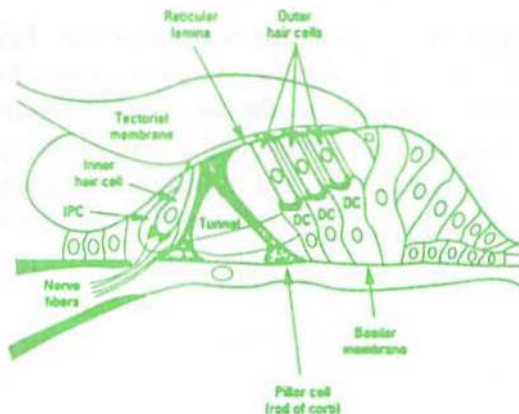


Figure 56 : The organ of Corti. DC = Deiter's cells (outer phalangeal cells).
IPC = inner phalangeal cells.

The hairs pierce a tough membrane called the *reticular lamina* which is supported by the *rods of Corti*. A thin viscous (but elastic) membrane known as the *tectorial membrane* covers the rows of hair cells, and in it the tips of the hairs are embedded (particularly those of the outer hair cells).

Within the modiolus, there are certain ganglia called the *spiral ganglia* (figure 54) which contain the nerve cell bodies of the cochlear nerve. These cells are *bipolar*, the peripheral branches of which (i.e. their dendrites) arborize extensively around the hair cells of the organ of Corti while their central branches (i.e. their axons) form the afferent fibres of the cochlear nerve within the modiolus.



Figure 57 : Sound waves produced by a vibrating tuning fork.

PHYSICS OF SOUND

Sound is produced by setting off its source (e.g. a tuning fork in figure 57) into a *vibrational motion*. Such vibrations should be transmitted through a medium (as air or water) to be perceived and heard by the ear. In air, sound

is produced as a result of vibration of the air molecules and is transmitted in ***the form of longitudinal waves*** i.e. as alternate phases of compression and decompression (or rarefaction) of the air molecules (figure 57). These waves travel in air at a speed of about ***344 meters / second*** (at a temperature of 20 °C in the sea level), and it increases with both temperature and altitude. In fresh water, sound is transmitted at a speed of about ***1450 meters/second***.

Sound is the sensation produced when sound waves strike the tympanic membrane. On the other hand, hearing is the ability to perceive and interpret the sound waves. The ear functions as a ***transducer***, because it transforms the pressure energy of the sound waves into nerve impulses, and the auditory cortex converts these impulses into the sensation of sound.

Properties (characteristics) of sound waves

(1) Frequency

This is the rate at which the air waves oscillate per unit time. It is expressed in ***Hertz (Hz) i.e. the number of cycles (waves) per second***, and it determines the ***pitch of sound*** (high frequencies produce high-pitched sounds and vice versa).

The human ear can discriminate frequencies (i.e. pitches) ranging from ***20 to 20000 Hz*** (best in the range of 1000-3000 Hz). Some animals (e.g. dogs and bats) can discriminate much higher frequencies.

During an ordinary conversation, the pitch of a male voice is about ***120 Hz*** while that of a female voice is about ***250 Hz***. A normal average individual can distinguish as much as ***2000 different pitches*** but trained musicians can discriminate a much greater number of pitches.

(2) Amplitude

The amplitude of sound waves determines ***the intensity or volume of the sound*** (i.e. its loudness or audibility). The greater the amplitude, the louder will be the sound and vice versa. ***However, the loudness of a certain sound is not only determined by the wave amplitude but also by its frequency*** e.g. a high pitched sound is heard louder than another sound having the same amplitude but is low-pitched (compare A & C in figure 58).

In practice, sound intensity is measured in ***bels (B) or decibels (dB)***. One B = 10 dB (i.e. one dB = 1/10 B).

The intensity of a certain sound in bels equals ***the logarithm of the ratio of the intensity of the sound to that of a standard sound*** i.e. :

The sound intensity in bels = $\log \frac{\text{intensity of the given sound}}{\text{intensity of the standard sound}}$

The standard sound is a sound having *a frequency of 1000 Hz and an intensity that is just audible in a young adult* (thus representing the *hearing threshold*). Accordingly, a sound which is just audible to the human ear (i.e. equal to the standard sound) would have an intensity of zero bels (because $\log 1 = \text{zero}$). *Therefore, a value of zero bels (or dB) does not mean absence of sound but a sound having an intensity equal to that of the standard.* On the other hand, in case of a whispering sound, the ratio of the intensity of the whisper to that of the standard sound is 100, therefore :

The intensity of the whisper = $\log 100 (10^2) = 2 \text{ bels} = 20 \text{ dB}$.

During ordinary conversations, the intensity of the sound is about 50 dB (5 bels) i.e. $10^5 (=100000)$ times greater in intensity than the standard sound.

Sound intensities of about **120 dB produce discomfort** while those of **140 dB are painful** and those of **160 dB (as those produced by a jet plane) have a very high energy** (they increase the body temperature and may cause damage to the delicate tissues of the ear).

Higher sound intensities (specially at high frequencies) lead to tissue destruction e.g. *the ultrasonic waves* (which have a frequency of 1000000 Hz). These waves are *inaudible and are clinically used to destroy localized pathological masses* (e.g. a localized brain tumour) without serious damage to the intervening tissues.

(3) Wave form

Different sound wave forms are perceived as a difference in the **quality (timbre) of the sound**. Simple sound waves (figure 58 A, B and C) produce sounds with **pure tones**. Sound waves that have **repeating patterns** even though the individual waves are complex (figure 58 D) are perceived as **musical sounds**. Most musical sounds are made up of a wave with a primary frequency (which determines its pitch) plus a number of harmonic vibrations (= overtones) that give the sound its characteristic timbre. On the other hand, **aperiodic non-repeating vibrations** (i.e. wave forms that have no regular repeated pattern) cause **a sensation of noise** (figure 58 E).

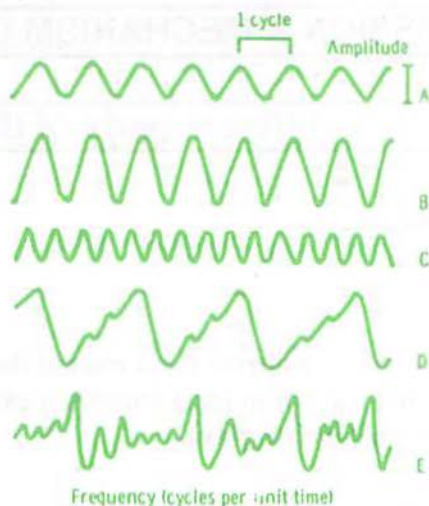


Figure 58 : Different patterns of sound waves.

- (A) Pure tone. (B) Pure tone, same frequency as A but greater amplitude (louder). (C) Pure tone, same amplitude as A, but greater frequency, so higher pitch (and also louder). (D) Complex wave form that is regularly repeated (perceived as a musical sound). (E) Waves having no regular pattern (perceived as noise).

MASKING

The presence of one sound decreases the ability of the ear to hear other sounds. This phenomenon is known as masking, and it is believed to be due to *relative or absolute refractoriness of the previously-stimulated auditory receptors and nerve fibres to other stimuli*. The degree to which a given tone masks other tones is *related to its pitch* (the higher the pitch, the greater will be its masking effect). The masking effect of noise definitely *raises the auditory threshold (i.e. decreases the auditory acuity)*.

CHAPTER 2

SOUND TRANSMISSION & MECHANISM OF HEARING

Functions of the different parts of the ear

(A) The external ear

(1) The pinna

This collects sound waves and directs them toward the external auditory meatus. In man, it is of little use but in most animals it can move reflexly to seek the source of sound (= *rotation reflex*).

(2) The external auditory meatus

This is a narrow canal about 25 mm long that performs the following functions :

- a) It is the passage through which sound waves reach the middle ear.
- b) Its skin lining secretes a *waxy material* which lubricates the drum.
- c) It contains *hair which traps foreign bodies* and, because it is narrow, it also prevents entrance of large particles (e.g. insects).

(3) The tympanic membrane (ear drum)

This is a thin ellipsoid diaphragm-like membrane that separates the external ear from the middle ear. Its surface area is about 55 mm^2 and its thickness is about 0.1 mm. It is conical in shape with its concave surface facing outwards and downwards (figure 53). Its central part is called the *umbo*, and the handle of malleus (the first bony ossicle) is attached to this part from inside (figure 60).

The drum moves in and out in response to the pressure changes produced by the sound waves. Therefore, it functions as *a resonator* that *reproduces the vibrations of the sound source*, and the amplitude of the vibrations is directly proportional to the intensity of the applied sound. On the other hand, the drum is *nearly critically damped* (i.e. its vibrations stop almost immediately when the sound waves applied to the ear cease).

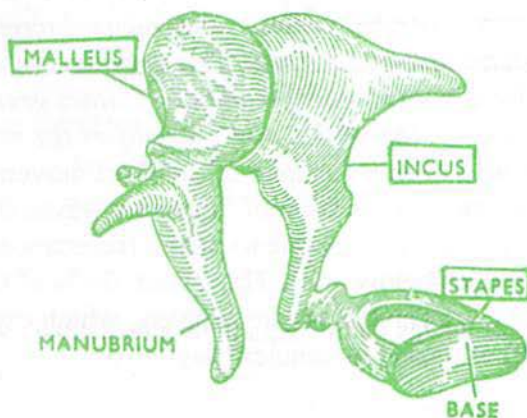


Figure 59 : The 3 bony ossicles of the middle ear

(B) The middle ear

(1) The bony ossicles

These include (from outside inwards) the *malleus (hammer)*, *incus (anvil)* and *stapes (stirrup)*. They constitute a chain connecting the tympanic membrane to the membrane covering the oval window. The handle (= manubrium) of malleus is attached to the inner side of the drum at the umbo (see above) while its head is attached to the wall of the middle ear, and its short process is attached to the incus. The long process of the incus passes downwards parallel to the handle of the malleus and articulates with the head of the stapes (figure 59). The *oval base (= footplate) of the stapes fits into the oval window* (figure 60), where it is attached to the margins of this window by a ligament called the *annular ligament*.

The vibrations of the drum are transmitted to the handle of the malleus, which rocks on an axis passing through the junction of its long & short processes, so that the short process transmits the vibrations to the incus. The incus moves in such a way that the vibrations are transmitted to the head of the stapes (figure 60), the movements of which swing its footplate to and fro like a door hinged at the posterior edge of the oval window.

Functions of the bony ossicles

The bony ossicles act as a *lever system* which performs the following functions :

a) Conversion of the resonant vibrations of the drum into movements of the stapes against the perilymph in the scala vestibuli.

b) The lever action of the malleus and incus *increases the force of movement of the stapes about 1.3 times*. This together with the fact that the *surface area of the drum (55 mm^2) is about 17 times greater than that of the foot plate of stapes (about 3.2 mm^2) amplifies the sound pressure at the oval window about 22 times* (in spite of the short movement of stapes).

Normally, during transmission of the sound waves in the middle ear, there is *some loss of sound energy* due to inertia (resistance) of the ossicles. However, at frequencies below 3000 Hz, about 60 % of the sound energy applied to the drum is transmitted to the cochlea, which explains why pitch discrimination is best at these frequencies (page 83).

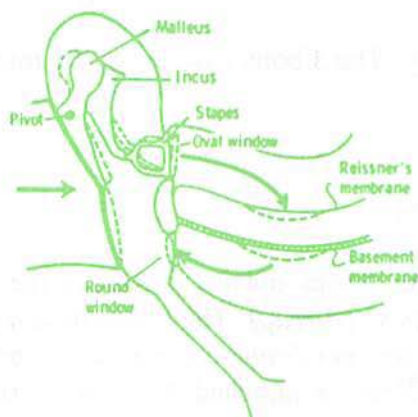


Figure 60 : Movements of the bony ossicles, as well as the vestibular and basilar membranes, in response to sound waves falling on the ear drum.

(2) Middle ear muscles

a) **Tensor tympani** : This muscle is attached to the malleus and on contraction, it pulls the handle of the malleus and the tympanic membrane inwards (increasing the stretch of the latter).

b) **Stapedius** : This muscle is attached to the stapes, and on contraction, it pulls the stapes outwards away from the oval window.

The tympanic reflex (= attenuation reflex)

This is reflex contraction of both the tensor tympani & stapedius muscles in response to excessively loud sounds. The simultaneous contraction of both muscles decreases sound transmission in the middle ear, which prevents excessive excitation of the auditory receptors, and protects the cochlea from injury by the strong sound vibrations. Such decrease in sound transmission

Section II – Chapter 2 Sound transmission & mechanism of hearing

is produced through :

a) The strong contraction of the tensor tympani causes excessive stretch of the tympanic membrane, which decreases its vibrations.

b) The simultaneous contraction of both muscles causes the entire ossicular system to be more rigid (which greatly reduces ossicular conduction).

However, the latent period (reaction time) of this reflex is only 40-80 (up to 160) milliseconds, so it cannot protect the inner ear from brief intense stimulations (e.g. that produced by a gunshot), because they will be transmitted to the inner ear before reflex contraction of these muscles occurs.

(3) The Eustachian tubes (auditory tubes)

These tubes connect the middle ears to the nasopharynx, and they are essential for *equalization of pressure at the 2 sides of the ear drums* (which is important for their resonator function). Normally, *they are kept closed at their pharyngeal ends* to prevent conduction of talking & breathing sounds to the middle ears (which decreases the auditory acuity). But, this would also decrease the auditory acuity because the air in the middle ears will be gradually absorbed, creating a negative pressure which would suck the eardrums inwards (leading to their excessive stretch and, consequently, reduction of their vibratory capacity). However, this does not normally occur because the tubes *temporarily open during swallowing, chewing and yawning*, allowing free communication between the middle ears and the nasopharynx which always re-equalizes the pressure at the 2 sides of the ear drums.

If the tubes are closed for a long time (e.g. as a result of common cold), the air in the middle ears will be gradually absorbed and the produced negative pressure sucks the eardrums inwards, leading to their excessive stretch, which decreases the auditory acuity (so the patient will temporarily suffer partial deafness).

During ascending in an airplane, the atmospheric pressure is decreased and the tubes open automatically, allowing air in the middle ear cavities to go out (thus decreasing the pressure within the middle ears, and an equal pressure is maintained at both sides of the ear drums).

However, during descending in an airplane, the atmospheric pressure increases and this *would lead to rupture of the ear drums* (which may occur in sleeping and unconscious individuals). Therefore, during the descent of airplanes, *one should swallow periodically* to open the Eustachian tubes, thus allowing air to enter the middle ears. In this way, the pressure inside the middle ears will also increase, and an equal pressure will be maintained at both sides of the eardrums (which prevents their rupture).

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(C) The internal ear (the cochlea)

This is the most important part in the ear because it contains the sense organ of hearing (*organ of Corti*), which is stimulated by sound vibrations transmitted from the middle ear through the oval window. Nerve impulses originating in the organ of Corti are transmitted to the auditory areas in the cerebral cortex via the auditory or cochlear (= 8th cranial or acoustic) nerve.

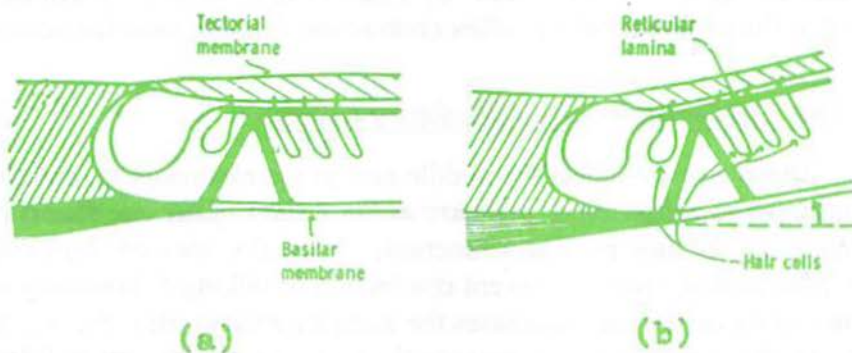


Figure 61: Position of the basilar membrane. (a) Normal resting position. (b) Upward displacement

MECHANISM OF HEARING

(1) The pinna collects sound waves and directs them toward the external auditory meatus (this function is minimal in humans).

(2) The sound waves cause vibrations of the tympanic membrane at the same frequency.

(3) The movements of the tympanic membrane are transmitted and amplified by the bony ossicles of the middle ear to the oval window (via the foot plate of the stapes).

(4) The movements of the footplate of the stapes lead to displacement of *the perilymph in the scala vestibuli*. The motion of this fluid moves the vestibular membrane which, through moving the endolymph in the scala media (figure 60), causes the basilar membrane to move in a pattern determined by the frequency and intensity of the sound waves.

A movement of the stapes inwards causes a downward displacement of the basilar membrane (figure 60) while a movement of the stapes outwards causes an upward displacement of the basilar membrane (figure 61 b) and in both conditions, a shearing motion occurs between the tectorial membrane

Section II – Chapter 2 Sound transmission & mechanism of hearing

and the reticular lamina (figure 56), leading to bending of the hair processes of the hair cells. This produces a receptor potential which initiates action potentials in the cochlear nerve.

Movements of the basilar membrane are maintained by changes in the round window. During downward displacement of the basilar membrane, the perilymph in the scala tympani is displaced leading to *bulging of the membranous covering of the round window outwards in the middle ear* (figure 60), and such bulging disappears during upward displacement of the membrane. If bulging of the round window is prevented, the pressure vibrations applied to the oval window will not cause movements in the basilar membrane (and consequently no stimulation of the auditory receptors).

The movement of perilymph in the scala vestibuli is not transferred to the scala tympani through the helicotrema (figure 55) because the transfer of such movement is so rapid that it cannot take place through this way.

Routes of sound wave transmission to the cochlea

(1) **The ossicular route** : This is the normal and ***most efficient route*** because it transmits the sound waves to the receptor cells of the inner ear without much loss of energy.

(2) **The bony route** : Sound waves can travel through the ***skull bones*** and reach the inner ear (e.g. by applying a vibrating tuning fork on the mastoid process). However, this route is ***less efficient*** than the previous one.

(3) **The air route** : The air vibrations in the middle ear are transmitted to the ***round window*** (so it can deliver some sound energy to the inner ear). However, this route is the ***least efficient*** because sound wave transmission directly from air to the perilymph is associated with much loss of energy.

Electric responses in the cochlea

Electrolyte distribution in the cochlea

Although the endolymph is an extracellular fluid, yet like intracellular fluids, its K^+ content is high (150 mmol /L) while its Na^+ content is low (1mmol /L), so it is the ***only extracellular fluid in the body with a high K^+ content*** (figure 62). The perilymph, on the other hand, as any other extracellular fluid, is rich in Na^+ (150 mmol /L) and poor in K^+ (3-5 mmol /L).

The ***perilymph is formed mainly from the plasma***, and there are small differences in its composition in the scalae vestibuli and tympani. On the

other hand, the *endolymph is formed by the stria vascularis* (figure 62), the cells of which have (a) A high concentration of Na^+ - K^+ ATPase (b) An electrogenic K^+ pump (which increases the K^+ content in the scala media and renders it electrically positive relative to the other 2 scalae).

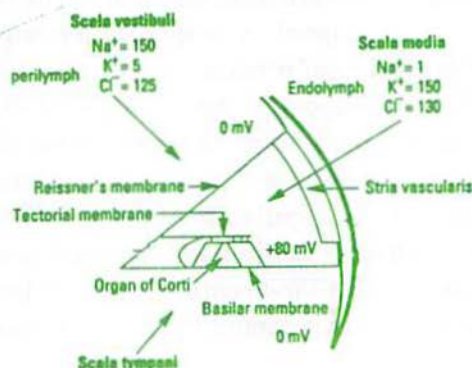


Figure 62 : The endocochlear potential. The endolymph is positive (+80 mV) relative to the perilymph (0 mV).

Electric phenomena in the cochlea

(1) The endocochlear (endolymphatic) potential

This is the *potential difference normally present between the endolymph and perilymph* (figure 62). It is about **80 millivolts**, the endolymph being *positive relative to the perilymph (0 mV)* and is produced and maintained by active processes that occur in the stria vascularis (see above).

(2) Cochlear microphonics (CM)

These are *potential changes that occur in the cochlea on exposing the ear to sound*. Such changes can be recorded by 2 electrodes (one applied at the cochlea and the other at an indifferent site). They have the same characteristics of the generator (receptor) potential (refer to C.N.S.) and are followed by action potentials in the cochlear nerve. They are produced as a result of **bending of the hair processes**, so their magnitude is proportionate to the degree of displacement of the basilar membrane (i.e. to the intensity of the applied sound).

The CM do not give information about the nature of the auditory sensation. However, their presence is a sign that the organ of Corti has been stimulated. Accordingly, recording of these potential changes is used in (a) Experimental studies of auditory functions (b) Localization of the sites of cochlear lesions.

CHAPTER 3

THE AUDITORY NERVOUS PATHWAY

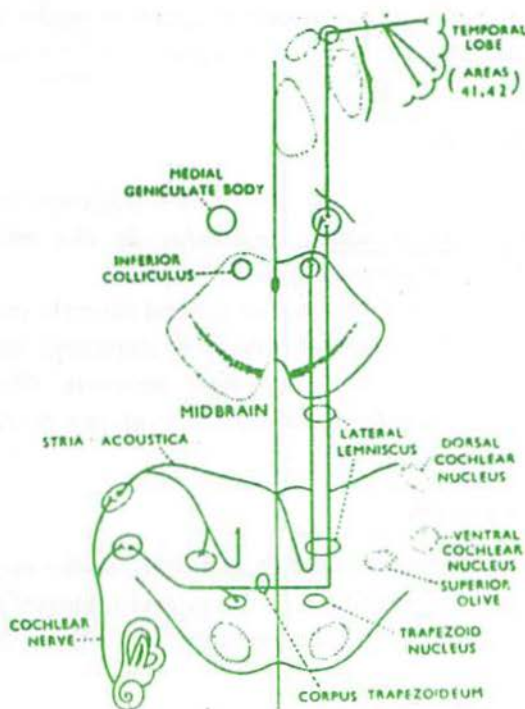


Figure 63 : The auditory nervous pathway

Nerve impulses are transmitted from the cochlea to the auditory areas in the cerebral cortex via **5 neurons** which include the following :

(1) First order neurons

These are afferent nerve fibres in the cochlear (8th cranial) nerve. They are *axons of the bipolar cells in the spiral ganglia* (figure 54), the peripheral branches of which transmit nerve impulses from the hair cells of the organ of Corti.

The cochlear nerve enters the brain stem at the upper border of the medulla oblongata, then it divides into 2 branches which *relay at the dorsal and ventral cochlear nuclei* (figure 63).

(2) Second order neurons

These arise from both cochlear nuclei and *relay in the superior olivary and trapezoid nuclei (= nuclei of the lateral lemniscus) at both sides*. The crossing fibres from the dorsal and ventral cochlear nuclei pass in *the stria acoustica and trapezoid body (= corpus trapezoideum)* respectively (fig. 63).

(3) Third order neurons

These arise from the superior olivary and trapezoid nuclei, ascend in *the lateral lemnisci of both sides, and relay in the midbrain at both inferior colliculi (= inferior corpora quadrigemina)*.

Some fibres from the cochlear nuclei ascend directly in the lateral lemnisci without relaying in the superior olivary & trapezoid nuclei. Therefore, *the lateral lemnisci contain both 2nd order neurons (from the cochlear nuclei) and 3rd order neurons (from the superior olivary & trapezoid nuclei)*

(4) Fourth order neurons

These arise from the inferior colliculi and *relay at the medial geniculate bodies of the thalamus*. Some fibres of the lateral lemnisci relay directly in the medial geniculate bodies without relaying in the inferior colliculi (fig. 63)

(5) Fifth order neurons

These arise from the medial geniculate bodies and pass via *the auditory radiations in the posterior limbs of internal capsules* (refer to C.N.S.) to reach their destination at the *auditory cortical areas in the temporal lobes of the cerebral cortex at both sides*. On each side, these areas include :

a. **The auditory sensory area (areas 41 & 42)** : This area is located in the upper part of the temporal lobe (mostly hidden in the floor of the lateral fissure). Its *anterolateral part receives impulses from the apex of the cochlea* (i.e. low pitched sounds), while *its posteromedial part receives impulses from the base of the cochlea* (i.e. high pitched sounds).

b. **The auditory psychic (= association or interpretative) area (area 22)** : This area lies adjacent to the auditory sensory area on the lateral surface of the superior temporal gyrus.

Functions of the auditory cortex

(A) The auditory sensory area is concerned with the following :

1) *Perception of the pitch, intensity and quality of sounds* (without understanding their meanings).

2) *Perception of the source of sound* : The localization of the source of sound depends on (a) The difference in time of arrival of the sound to both ears (b) The difference in loudness of the sound in both ears (the sound reaches faster & is louder in the ear which is closer to the source of sound) (c) An intact auditory sensory area. Thus the presence of both ears as well as an intact auditory cortex are essential for perception of the source of sound.

(B) The auditory psychic area : This interprets (explains and understands) the meaning of various sounds and words. Therefore, a lesion affecting this area on the left side (in right-handed individuals) leads to ***auditory aphasia or word deafness*** (refer to C.N.S.).

Functions of the inferior colliculus

From the inferior colliculus, the ***ventral tectospinal and tectobulbar tracts arise***. These tracts descend and terminate at the lower motor neurons mediating the **auditory spinal reflexes** (refer to C.N.S.) Therefore, the inferior colliculus performs the following functions :

- (1) It is the *centre of various auditory reflexes*.**
- (2) It is an *important centre in the auditory nervous pathway*** (see above).
- (3) The interconnection between the inferior colliculi of both sides allows *bilateral cortical representation of the auditory sensation*** (see below).

Localisation in the auditory pathway

As in the auditory cortex, there is a ***specific localization of the various parts of the cochlea throughout the auditory pathway***. Thus, selective destruction of minute portions of the medial geniculate body leads to deafness for only certain bands of frequencies.

Bilateral representation of the auditory sensation

The auditory impulses from each ear are bilaterally represented in both temporal lobes (i.e. ***they reach the cortical auditory areas at both sides***).

This is because they are transmitted *via the 2 lateral lemnisci* and, in addition, there are *interconnecting fibres between the inferior colliculi* of both sides (see above). Therefore, *unilateral cortical lesions affecting the auditory areas (or unilateral lesions in the posterior limb of the internal capsule) do not cause deafness in either ear but only reduction of the hearing power at both sides* (refer C.N.S.).

REMARKS

(1) In each cochlear nerve, there is a prominent bundle of cholinergic efferent nerve fibres known as the *olivocochlear bundle* (which arises from the superior olivary nuclei of both sides and ends primarily around the bases of the outer hair cells in the organ of Corti). This bundle is probably an *efferent inhibitory pathway to the cochlea*, and it is believed that it plays an important role in the *regulation of the sensory output from this organ*.

(2) 90-95 % of the afferent neurons in the cochlear nerve arise from the *inner hair cells of the organ of Corti* (and only 5-10 % of these neurons arise from the much more numerous outer hair cells).

(3) The afferent and efferent nerve fibres in *each cochlear nerve* are about *28000 fibres*.

(4) The *inner hair cells are the primary sensory cells that generate action potentials in the cochlear nerve* although they are probably not directly attached to the tectorial membrane (however, their hairs are bent by fluid moving between the tectorial membrane and the underlying hair cells).

(5) The *outer hair cells are motile* (they shorten when depolarized and elongate when hyperpolarized). They have little direct receptive function, but they *improve hearing by influencing the vibration patterns of the basilar membrane* (by an unknown mechanism).

CHAPTER 4

THEORIES OF HEARING

Many theories attempt to determine the site and mechanism of discrimination of the pitch of sounds, the commonest of which are the following :

(1) Place (or resonance) theory

Depending on the structure of the basilar membrane, this theory assumes that *discrimination of the sound frequency is a function of the cochlea and not the auditory cortex* (which only interprets various frequencies as different pitches).

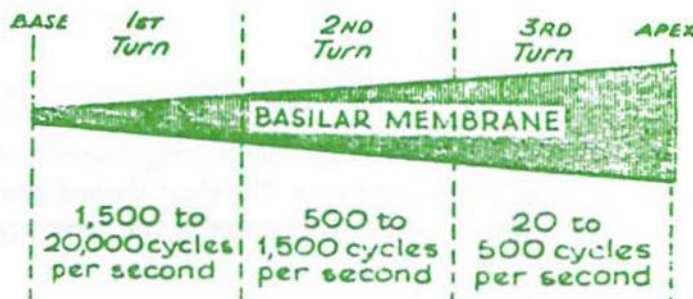


Figure 64 : The Basilar membrane. The frequencies that stimulate each part are indicated below the membrane.

The basilar membrane is suspended between the spiral lamina (the bony ridge protruding from the modiolus), and the bony wall of the cochlea. It *extends from the base to the apex of the cochlea in a spiral form* (turning round the modiolus), and on it is located the organ of Corti (which also has a spiral shape). Structurally, the basilar membrane is a *V-shaped fibrous membrane* (figure 64) that contains 20000-30000 elastic fibres embedded in a gelatinous material. It is about 32 mm long and is not under tension. At the base of the cochlea, the membrane is narrow (about 0.04 mm) and the fibres are stiff. On the other hand, at the apex of the cochlea, the membrane is much wider (about 0.5 mm) and the fibres are more lax.

The place theory postulates that the *fibres of the basilar membrane act as resonators and that each group of these fibres can be set into vibration only in response to a particular sound frequency*. When a group of fibres in the basilar membrane vibrates in response to a particular frequency, the organ of Corti at this part only is stimulated, while it remains silent at other parts, and the stimulated part discharges impulses in particular cochlear

nerve fibres to a specific area in the auditory cortex.

It was found that *high frequency sounds produce resonance in the short stiff fibres at the base of the cochlea, while low frequency sounds produce resonance in the long lax fibres at its apex* (figure 64).

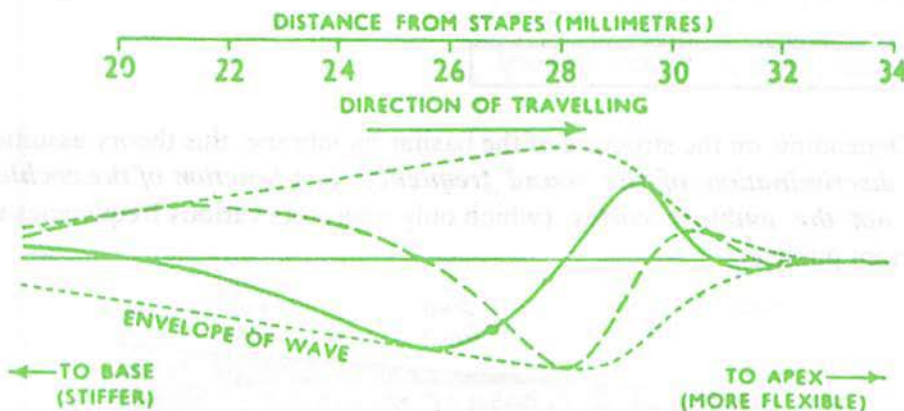


Figure 65 : Travelling wave theory. The solid & long-dashed lines represent the same sound wave at 2 different times. The short-dashed lines show the envelope of the wave (formed by connecting the wave peaks).

(2) Travelling wave theory

This is the *most accepted theory of hearing*. It is a modification of the place theory *since it also considers the cochlea to be the site of pitch analysis* (i.e. the frequency analyser of sound). It states that the movements of the foot plate of the stapes set up a series of travelling waves in the perilymph of the scala vestibuli, the height of which increases to a maximum then drops off rapidly (figure 65). The *basilar membrane is depressed into the scala tympani by the peaks of the waves in the scala vestibuli* (leading to stimulation of the organ of Corti at that part) and at the same time, the *round window bulges into the middle ear* (page 91).

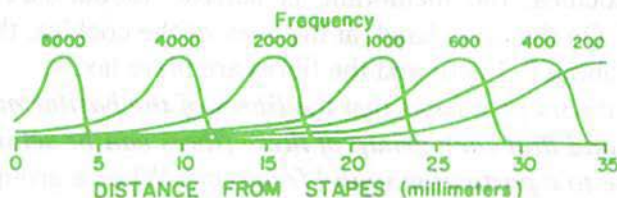


Figure 66 : Points of maximal displacement of the basilar membrane by waves generated as a result of stapes vibrations produced by sound frequencies between 200 and 8000 Hz.

It was found that the distance from the stapes to the point of maximum height of the waves (i.e. the point of maximum displacement of the basilar membrane) *varies with the frequency of sound waves* (figure 66). *High-frequency sounds generate waves that reach maximum heights near the base of the cochlea, while low-frequency sounds generate waves that reach maximum heights near its apex. Thus, high pitched sounds are perceived at the base while low pitched sounds are perceived at the apex of the cochlea.*

Discrimination of sound amplitude (intensity or loudness)

This is a function of the auditory cortex and it depends on the frequency of impulses discharged in the cochlear nerve to the cortical auditory areas. The latter is determined by the intensity of the sound. The greater the intensity of the sound, the more will be the displacement of the basilar membrane. This increases the shearing motion of the hair cells of the organ of Corti leading to greater cochlear microphonics (page 92) and consequently, more frequent discharge of impulses in the cochlear nerve. The increase in the number of the discharged impulses is interpreted by the auditory cortex as an increased intensity (or loudness) of sound. If the sound intensity is low, a reverse effect occurs.

CHAPTER 5

DEAFNESS AND HEARING TESTS

DEAFNESS

Deafness means loss of the hearing power, and it may be *complete or partial*. The latter condition is associated with rise of the auditory threshold. There are 2 main types of deafness :

(A) Conduction (conductive or transmission) deafness

This occurs due to *interference with the conduction of sound waves from the atmosphere to the cochlea*. Its main causes are :

- (1) *Obstruction of the external auditory meatus* e.g. by excessive wax, a foreign body or an inflammatory swelling (*otitis externa*).
- (2) *Damage or perforation of the tympanic membrane*.
- (3) *Infections of the middle ear* (= *otitis media*).
- (4) *Otosclerosis* (i.e. stiffening and immobilization of the bony ossicles).
- (5) *Blockage of the Eustachian tube* (commonly as a result of spread of infection from the throat). The air in the middle ear is absorbed creating a negative pressure that leads to inward bulging of the drum, which greatly reduces its vibratory capacity (page 89).

(B) Nerve (perceptive) deafness

This occurs due to *interference with the transmission of nerve impulses from the cochlea to the auditory cortex*. Its main causes are :

- (1) *Damage of the basilar membrane or the organ of Corti* (e.g. as a result of prolonged use of the antibiotic streptomycin).
- (2) *Damage of the cochlear nerve* (e.g. due to severe head injuries or certain brain tumours).
- (3) *Meniere's syndrome*, in which there is an increase in the pressure of the endolymph inside the scala media and bulging of its walls (refer to C.N.S.).
- (4) *Extensive lesions of the auditory nervous pathway* (specially if bilateral) : These may occur as a result of either severe head injuries or certain brain tumours and infections (= central deafness).

HEARING TESTS

(A) Tests that diagnose deafness

(1) **Watch test** : The examiner stands *behind the patient* and approaches a watch gradually towards the patient's ear, till he hears its ticks (each ear is examined separately). The distance at which the patient first hears the ticks of the watch is measured and compared with that of a normal individual.

(2) **Tuning fork test** : This is the same as the preceding test, but a vibrating tuning fork is used instead of the watch.

(3) **Audiometry** : The audiometer accurately diagnoses deafness as well as its type and degree (see below).

(B) Tests that differentiate between nerve & conduction deafness

(1) Rinne's test

The base of a vibrating tuning fork is placed on the **mastoid process** (figure 67 a) till its vibrations are no longer heard, then its prongs (limbs) are brought close to the ear (figure 67 b). Normally, the vibrations will still be heard for about 45 seconds (*+ve test*). This is because **normally air conduction of sound waves is better than bone conduction**. If there is conduction deafness, no vibrations will be heard after shifting the fork close to the ear (*-ve test*) because in this condition bone conduction is better than air conduction. On the other hand, if there is nerve deafness, both bone and air conduction are impaired (so the patient's hearing power is much reduced whether the fork is placed on the mastoid process or applied close to the ear). *However, in some cases of nerve deafness (specially if partial) the Rinne's test may be positive.*

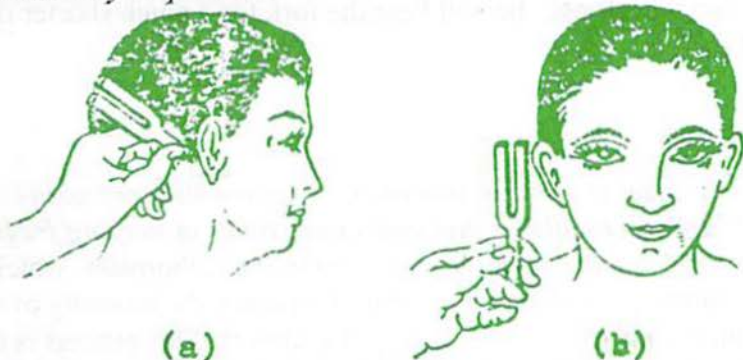


Figure 67 : Rinne's test



Figure 68 : Weber's test

(2) Weber's test

The base of a vibrating tuning fork is placed *on the middle of the forehead* (figure 68). Normally, its vibrations are heard equally at both ears. If there is *conduction deafness in one ear, the vibrations will be louder in that ear than in the healthy ear because the masking effect of the environmental noise is absent in the deaf ear* (but present in the healthy ear). On the other hand, if there is nerve deafness in one ear, the vibrations will not be heard in that ear and will be louder in the healthy ear.

(3) Schwabach's test

This test is simply *a comparison of bone conduction in the patient with that in a normal subject* (by placing the vibrating tuning fork on the mastoid process). *If the patient has conduction deafness, he will hear the tuning fork for a longer time than the normal subject*. On the other hand, if he has nerve deafness, he will hear the fork for a much shorter time than the normal subject.

(4) Audiometry

The audiometer is an apparatus used for testing auditory acuity. Simply, it is an *electronic oscillator that emits pure tones of varying frequencies*. The subject puts on an earphone connected to the audiometer, which is then set up to emit a pure sound at a certain frequency the intensity of which is gradually increased till it is heard by the subject. This process is repeated using other normally audible frequencies, and the results are recorded on a

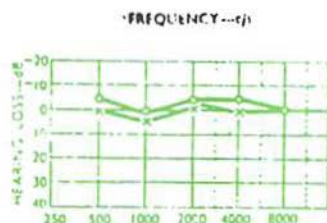


Figure 69 : A normal audiogram. o = right ear. x = left ear.

special chart called the **audiogram** (figure 69).

The apparatus is calibrated so that the normal threshold intensity for each tone is indicated by zero decibel (dB) loss. Positive losses (below the zero line) indicate impairment of the auditory acuity, while negative losses (above the zero line) indicate a better auditory acuity than the average normal (figure 69).

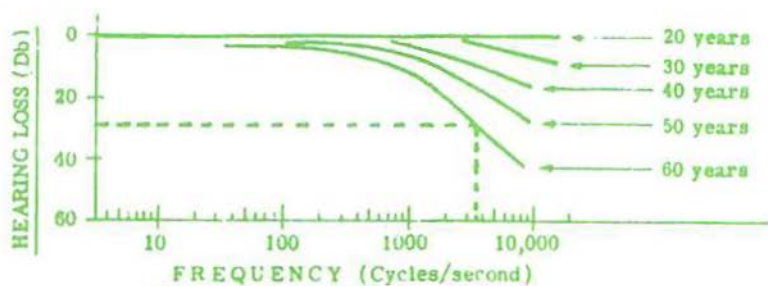


Figure 70 : Audiograms at various ages.

Auditory acuity

This can be estimated by **determining the threshold of audibility** (= lowest sound intensity which gives a sound sensation). Normally, the auditory acuity decreases with advance of age, specially for the high frequencies of sound. Thus, at the age of 60 years, there is often a loss of hearing amounting to about 30 decibels (3 bels) at a frequency of 7000 Hz (figure 70). This means that the intensity of a sound at that frequency should be increased 1000 times than normal in order to be heard. This phenomenon is known as **presbycusis** (= old age hearing), and it is probably due to a decrease in the elasticity of different parts of the ear that occurs in old age.

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SECTION III

THE CHEMICAL SENSES

(1) *PHYSIOLOGY OF SMELL* (*OLFACTION*)

The sense of smell is important for the following :

- (1) Selection of the type of food.
- (2) Initiation of various secretions in the GIT through conditioned reflexes.
- (3) Determination of the flavor of food (with taste).
- (4) Control of sexual functions in animals and probably in man (it is more acute in women particularly at the time of ovulation).

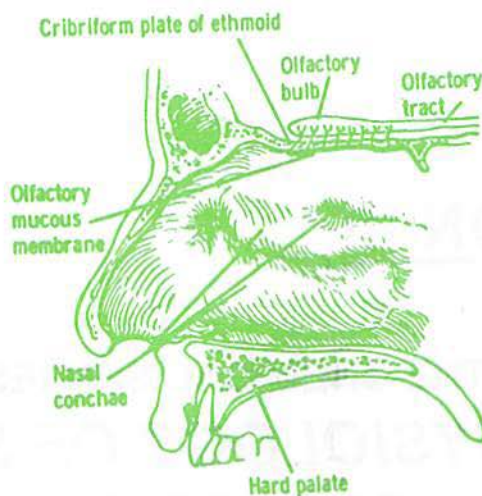


Figure 71 : The olfactory mucous membrane at the roof of the nasal cavity.

The olfactory receptors

These are distant receptors (teleceptors) which together with the taste receptors constitute the *external chemoreceptors* (so both sensations are called the *chemical senses*). The olfactory receptors are located in a special part of the nasal mucosa called the *olfactory mucous membrane* which is yellowish in colour and occupies an area about 5 cm^2 at the roof of the nasal cavity near the nasal septum (figure 71). In many animals e.g. dogs, the smell sensation is much more acute than in man, and their olfactory mucous membranes are much larger (so they are called *macrosmatic animals*). In comparison, *man is microsmatic*.

The olfactory mucous membrane is *constantly covered by mucus* (which is secreted by Bowman's glands) and it contains *3 main types of cells*, which include the following (figure 72) :

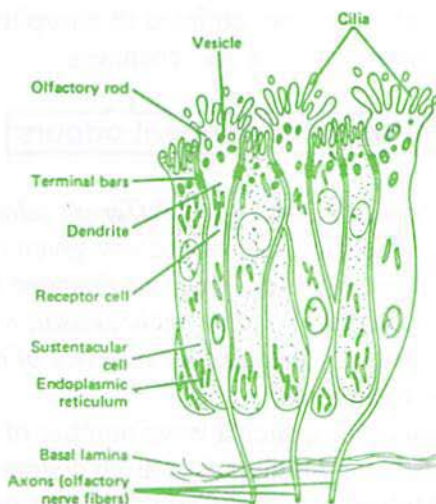


Figure 72 : Histology of the olfactory mucous membrane.

(1) Supporting (sustentacular) cells.

(2) Neuroblast like progenitor cells (which form the olfactory receptor neurons)

(3) Olfactory receptor cells : These are *actual bipolar neurons* (about 10 - 20 million in number) having short thick dendrites with expanded ends called the *olfactory rods* from which *cilia* project to the surface of the mucus. Their axons pierce the *cribriform plate of the ethmoid bone* and enter the *olfactory bulb* (figure 71).

Stimulation of the olfactory receptors

These receptors respond only to substances that *are in contact with the olfactory epithelium and are dissolved in the thin layer of mucus* that covers it. Therefore, odoriferous substances (odorants) should be (a) Volatile (b) Relatively highly water and lipid-soluble.

Mechanism of olfactory receptor stimulation

The overlying mucus contains one or more *odorant-binding proteins (OBP)* that concentrate the odorants and transfer them to the olfactory receptors leading to their activation. This occurs by activation of the adenylyl cyclase enzyme (via a G protein) which leads to increased formation of *cyclic AMP*. The latter binds to and opens Na^+ channels resulting in depolarization (i.e. receptor potential) which initiates action potentials i.e. nerve im-

Section III***Physiology of smell (olfaction)***

pulses. Some odorants may be coupled to phospholipase C, and the produced *inositol phosphates* open the Na^+ channels.

Power of discrimination of different odours.

Man can distinguish **2000-4000 different odours**. However, discrimination of the differences in intensity of any given odour is *poor*. The concentration of an odorant substance must be changed by about 30 % before a difference can be detected. *So the smell sensation in man is concerned mainly with the detection of presence or absence of odours rather than with the detection of their intensities.*

The discrimination of such a large number of different odours by the simple olfactory system (which lacks a high degree of complexity) may be explained by (a) Multiplicity of different odorant receptors (b) Analysis of the incoming signals in the brain (c) Both a & b combined. Recently, it was also suggested that *different olfactory glomeruli* (see below) respond to different odours (thus, *it is possible that these glomeruli are the real clue to the analysis of different odour signals transmitted to the C.N.S.*).

Olfactory adaptation

The smell sensation shows *fairly-rapid adaptation* (within a few minutes). So when an individual is exposed to a certain odour (even the most disagreeable ones), the perception of the odour gradually decreases and finally disappears. The *adaptation is specific* i.e. it occurs for this odour only (while other odours are perceived normally).

Olfactory adaptation is *primarily central in origin*, and is minimally due to adaptation of the olfactory receptors themselves, but its mechanism is not settled. However, it was found that stimulation of efferent fibres in the olfactory striae decreases the electric activity of the olfactory bulb, probably due to stimulation of certain centrifugal fibres (see below). Accordingly, it is suggested that after perception of smell in the C.N.S., *a feedback inhibition to the olfactory bulb activity occurs via certain centrifugal fibres* leading to decreased discharge of smell signals (i.e. adaptation to the odour).

Sniffing

Normally, the region of the olfactory receptors is *poorly ventilated* (air reaches it only through *eddy currents* produced by convection when cool air

Section III

Physiology of smell (olfaction)

strikes the warm mucosal surface). Sniffing is an action which includes contraction of the lower part of the nares on the nasal septum to help deflection of the air stream upwards. It is a *semi-reflex response* that usually occurs when a new odour attracts attention.

Pain nerve endings in the olfactory membrane

These are naked endings that discharge in the *trigeminal nerve*. They are stimulated by *irritant substances* (e.g. ammonia, chlorine, menthol and pepper). They are *also concerned with initiating sneezing, lacrimation, respiratory inhibition and other responses to nasal irritants*.

Tests of smell sensation

- (1) The subject *sniffs several common odorants* (which must not be irritant) and is asked to differentiate between them. *Each nostril should be examined separately*.
- (2) **Olfactometry** : This is investigation of the smell threshold by a special instrument called the *olfactometer*.

Abnormalities of smell sensation

Smell may be lost (*anosmia*), diminished (*hyposmia*) or distorted (*dysosmia*). Different types of anosmias are detected in humans, and each is probably due to congenital absence (or disturbed function) of one of the odorant receptors. *In old age, the sense of smell is normally decreased* (so its threshold increases with advancing age).

Detection of the source of odour

Similar to the detection of the source of sound (page 95), the detection of the source of an odour *depends upon the slight difference in the time of arrival of the odorant molecules to the 2 nostrils*. These molecules come faster (and in a slightly greater concentration) to the nostril close to the source of the odour. *Therefore, the presence of the 2 nostrils is required for detection of the source of an odour (in addition to an intact olfactory nervous pathway)*.

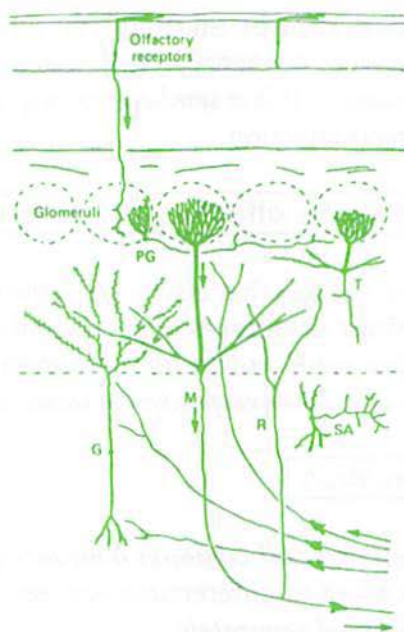


Figure 73 : Neural elements in the olfactory bulb. M = Mitral cell. T= Tufted cell. G = Granule cell. PG = Periglomerular short axon cell. SA = Deep short axon cell. R = Recurrent collateral from mitral cell axon. Notice the efferent output (the mitral cell axons) and the 3 centrifugal afferent inputs.

The olfactory nervous pathway

The axons of the olfactory receptor neurons pierce the cribriform plate of the ethmoid bone and **enter the olfactory bulb** where they terminate in relation to the dendrites of 3 types of cells (the **mitral cells**, **tufted cells** and **periglomerular short axon cells**) forming complex globular synapses called **olfactory glomeruli** (figure 73). Each olfactory bulb contains several thousands of such glomeruli, and an average of 26000 receptor cell axons converge on each glomerulus. Other cells in the olfactory bulb include the **granule cells** (the role of which is **mostly inhibitory**) and the **deep short axon cells** (the function of which is unknown).

The output (efferent) fibres that leave the olfactory bulb via the **olfactory tract** (figure 71) are **mostly axons of the mitral cells** (figure 73). These axons pass posteriorly through the **intermediate and lateral olfactory striae** and **terminate on the dendrites of pyramidal cells in the olfactory cortex**. The latter includes the following structures (which are part of the **limbic system**) :

Section III**Physiology of smell (olfaction)**

(1) **The anterior olfactory nucleus** : This is concerned with coordination of input from the contralateral olfactory cortex, and possibly transfer of olfactory memories from one side to the other.

(2) **The piriform cortex** : This is concerned with olfactory discrimination and probably also its conscious perception (*the main olfactory area*).

(3) **The corticomedial part of the amygdaloid nucleus** : This is probably concerned with the emotional responses to olfactory stimuli.

(4) **The entorhinal cortex** : This is probably concerned with olfactory memories.

****** It is noticed that, unlike all other sensations, **no fibres in the olfactory nervous pathway pass through the thalamus** (so, olfaction is the only sensation in the body that does not pass through the thalamus during its course to higher cortical centres).

The olfactory bulb also receives 3 afferent inputs (figure 73) in addition to that received from the olfactory mucous membrane, from the following areas :

- a. The nucleus of the diagonal band (= ***centrifugal fibres***).
- b. The ipsilateral anterior olfactory nucleus.
- c. The contralateral anterior olfactory nucleus (which reaches the olfactory bulb via the anterior commissure).

The centrifugal fibres as well as the other 2 afferent inputs (which also pass centrifugally in the olfactory tract i.e. ***in a backward direction from certain brain areas to the olfactory bulb***) terminate mostly at ***the granule cells*** (figure 73). These cells, in turn, inhibit other cells in the olfactory bulb (specially the mitral and tufted cells). Such inhibitory feedback pathway may be a means to sharpen the excitation within the olfactory bulb (which increases the ability to distinguish one odour from another), and may also explain ***how central olfactory adaptation takes place*** (page 10).

1. The first part of the report is a general introduction to the subject.

2. The second part is a detailed description of the methods used.

3. The third part is a discussion of the results obtained.

4. The fourth part is a conclusion and a summary of the findings.

5. The fifth part is a list of references.

6. The sixth part is a list of figures and tables.

7. The seventh part is a list of appendices.

8. The eighth part is a list of footnotes.

9. The ninth part is a list of acknowledgments.

10. The tenth part is a list of the author's address and contact information.

11. The eleventh part is a list of the author's previous work.

12. The twelfth part is a list of the author's future work.

13. The thirteenth part is a list of the author's publications.

14. The fourteenth part is a list of the author's awards and honors.

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SECTION IV

THE CHEMICAL SENSES (2) *PHYSIOLOGY OF TASTE* (*GUSTATION*)

The sense of taste is important for the following :

- (1) Initiation of various secretions in the GIT through **unconditioned reflexes** (refer to digestion).
- (2) Determination of the **flavor of food** (with smell).
- (3) **Prevention of ingestion of most poisons** (which usually have a bitter taste because most of them are alkaloids).

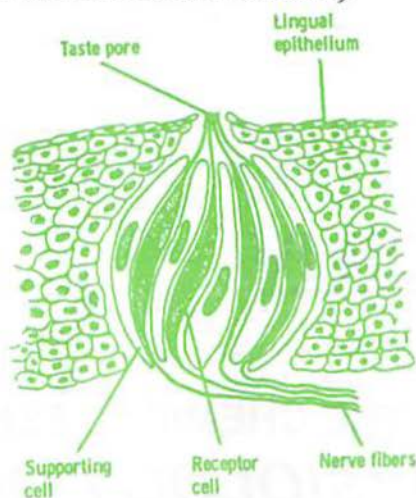


Figure 74 : Structure of a taste bud.

The taste receptors

These are the **taste buds** (figure 74) which are external chemoreceptors (with smell) and are about **10000 in adults** (but many of them rapidly degenerate after the age of 45 years).

Each taste bud is an ovoid body formed of **4 types of cells** which are (a) Basal cells (b) Type 1 cells (c) Type 2 cells (d) Type 3 cells. Both types **1 and 2 cells are sustentacular cells**, while **type 3 cells are the gustatory receptor cells that make synaptic connections with the sensory nerve fibres**. The type 1, 2 and 3 cells have **microvilli** that project into the taste pore. The basal cells are formed from the surrounding epithelium, and they **can differentiate into receptor cells** (the old receptor cells are continuously replaced every about 10 days).

When a sensory nerve is cut, the corresponding taste buds degenerate, but if the nerve regenerates, the neighbouring cells become organized into new buds. Each bud is innervated by about **50 nerve fibres**, and they are located in the mucosa of the epiglottis, palate and pharynx as well as in the **fungiform and circumvallate papillae of the tongue** (the filiform papillae

do not usually contain taste buds). There are about 5 buds /fungiform papilla (at its top) and 100 buds /circumvallate papilla (along its sides). The former are located near the tip of the tongue while the latter are arranged in a V-shaped area at the back of the tongue (figure 75).

Stimulation of the taste buds

These chemoreceptors respond only to the *dissolved substances in the oral fluids bathing them* (e.g. the saliva). The degree of taste depends on the solubility (thus the greater the solubility of a substance, the more it stimulates the taste buds and the greater the number of impulses transmitted to the cerebral cortex and, accordingly, the greater will be its degree).

However, *binding of the substances to the taste buds is weak* [since a little amount of water or saliva can abolish the taste sensation].

Stimulation of the taste buds can occur from *inside the body* (e.g. the bitter taste felt in jaundice is produced by the circulating bile salts).

As food passes through the buccal cavity, its volatile part is exhaled into the nasal cavities where it is smelled. Because *olfaction is more acute than taste in determining the flavor of food*, a patient having a common cold complains loss of taste (although he actually has lost smell).

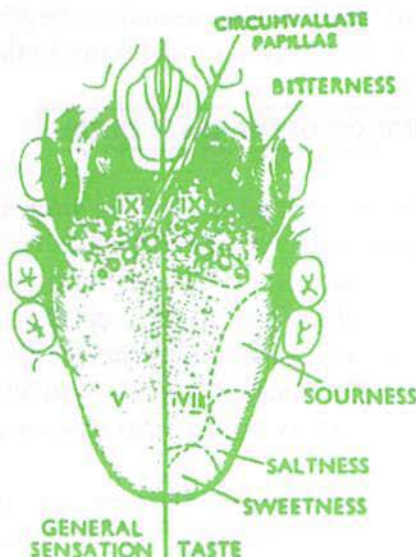


Figure 75 : Distribution of different taste modalities on the tongue. Also shown are the sensory nerves subserving taste sensation (right) and the sensory nerves subserving general sensations (left).

Taste modalities (= types of taste sensations)

In man, there are **4 basic taste modalities** (figure 75) :

(1) **Bitter** : This is tasted at *the back of the tongue* e.g. by quinine sulphate, strychnine hydrochloride, morphine, nicotine, caffeine and urea, as well as the inorganic salts of magnesium, ammonium and calcium (where the bitter taste is produced by the cation). Bitter taste is mostly sensed by the *circumvallate papillae*.

(2) **Sour** : This is tasted along *the edges of the tongue* e.g. by the H^+ ions of acids (for any given acid, the degree of sourness is directly proportional to its H^+ ion concentration). Organic acids cause more sour taste than inorganic acids (probably because they penetrate the cells more rapidly).

(3) **Sweet** : This is tasted at *the tip of the tongue* e.g. by sucrose, maltose, lactose, glucose, glycerol, ketones, polysaccharides, some alcohols, chloroform and *saccharin*. The latter produces satisfactory sweetening in very small amounts compared with sucrose (*so it is commonly used by both diabetic patients and obese persons for reducing the body weight*). Lead salts also produce a sweet taste.

(4) **Salt** : This is tasted at *the dorsum of the tongue anteriorly* by some cations of inorganic salts specially Na^+ , (but some organic compounds also taste salty).

The 4 modalities of taste are also sensed in the *pharynx and epiglottis* as well as in *the palate* (particularly sour and bitter in the latter site).

Mechanism of stimulation of the taste buds

The taste substances act on the exposed *microvilli* in the taste pores to produce generator potentials in the receptor cells, which initiate action potentials in the sensory neurons. A protein that binds, concentrates and transmits taste-producing substances to the receptor cells is secreted by Ebner's glands on the dorsum of the tongue, *acting like the OBP described in olfaction* (page 105). The mechanism of production of the generator potential varies from one gustatory modality to another as follows :

(1) *Sweet substances* increase the intracellular content of cyclic AMP (by activating the adenyl cyclase enzyme), which reduces K^+ conductance, resulting in production of the generator potential.

(2) *Bitter substances* produce the generator potential by triggering release of Ca^{++} from the endoplasmic reticulum.

Section IV***Physiology of taste (gustation)***

(3) ***Salty substances*** produce the generator potential by inducing Na^+ influx through passive ungated channels in the receptor's membrane.

(4) ***Sour substances*** produce the generator potential by blocking the K^+ channels in the receptor's membrane.

Specificity of the taste receptors

The taste buds are not histologically different in the different areas. However, recent studies showed that *some taste buds respond best to bitter stimuli whereas others respond best to salt, sweet or sour stimuli.*

Adaptation of taste receptors

As in case of the olfactory sensation, adaptation to taste rapidly occurs and is *also primarily central in origin* (but its mechanism is unknown).

Taste preference

This is the selection of certain types of food in preference to others. It is *more apparent in animals*, and it helps to control the type of diet they eat according to the body needs *e.g. adrenalectomized animals automatically select drinking water with a high NaCl concentration in preference to pure water.* Similarly, *parathyroidectomized animals prefer drinking water with high Ca^{++} concentration, and hypoglycaemic animals select the sweetest food from many samples.* The exact mechanism of this phenomenon is unknown (although it is almost certain that the C.N.S. is involved).

Taste abnormalities

These include *ageusia or taste blindness* (absence of the sense of taste), *hypogeusia* (diminished taste sensitivity) and *dysgeusia* (disturbed sense of taste). These may occur in certain diseases and also by many drugs *e.g. captopril* (which causes temporary unexplainable loss of taste sensation).

Discrimination of taste

As in case of the olfactory sensation, the ability to discriminate differences in the intensity of taste in man is *poor*. A 30% change in the concent-

ration of the tasted substance is also necessary before a difference in its intensity can be detected.

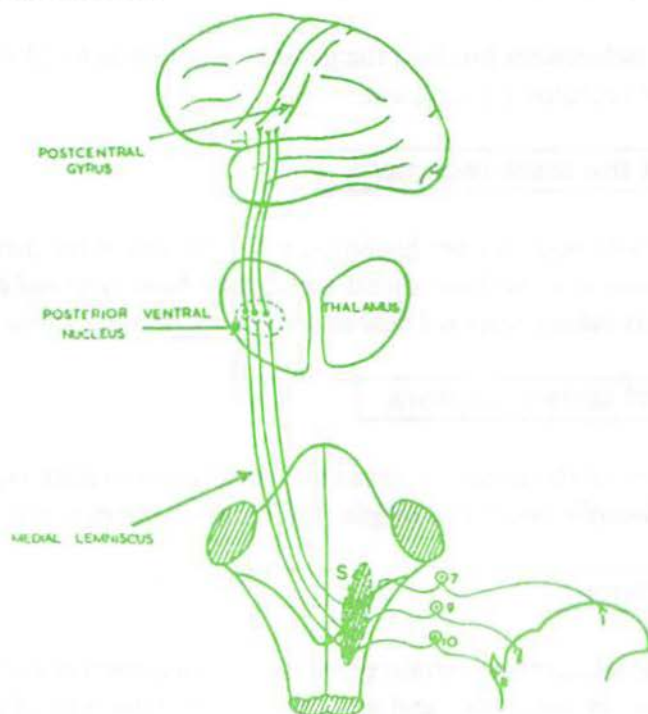


Figure 76 : Nervous pathway of the taste sensation. S = Nucleus of tractus solitarius.

Nervous pathway of taste

The sensory nerve fibres that arise from the taste buds are myelinated (but slowly conducting) and constitute the **first order neurons** in the nervous pathway of taste sensation. Those arising from the anterior 1/3 of the tongue travel in **the chorda tympani** (branch of the 7th cranial nerve) while those arising from the posterior 1/3 of the tongue travel via the **glossopharyngeal nerve** (9th cranial nerve). On the other hand, the sensory taste fibres arising from the pharynx and epiglottis are transmitted by **the vagus nerve** (10th cranial nerve).

These sensory fibres unite in the medulla oblongata forming the **tractus solitarius**, which relays in the **nucleus of the tractus solitarius**. **Second order neurons** start at this nucleus, cross the midline, join the **medial lemniscus** and ascend till terminating at the **posterior ventral nucleus of the thalamus** (figure 76). **Third order neurons** arise from the

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thalamus, pass in the sensory radiation (via the posterior limb of the internal capsule) and terminate at the *taste projection area of the cerebral cortex in the foot (i.e. lower part) of the postcentral gyrus*. This area is not specific for taste sensation, but it also subserves sensations from the face as well as other sensations from the tongue (see below).

****** Other sensations from the tongue include touch, heat, cold and pain sensations. They are transmitted from the anterior 2/3 of the tongue by the sensory fibres of the trigeminal nerve (5th cranial nerve) and from its posterior 1/3 by the sensory fibres of the glossopharyngeal nerve (9th cranial nerve) as shown in figure (75).

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